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CORONARY ARTERIOGRAPHY*

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VISUALIZATION of the blood supply to any organ can provide information of considerable clinical importance. It is well established that such procedures as cerebral angiography, angiocardiography, thoracic and abdominal aortography, peripheral arteriography and venography are indispensable in the armamentarium of medical practice. Indeed, the medical literature attests to progressive efforts toward attainment of selective roentgen visualization of the vascular supply of almost every organ and/or organ system. While there have been numerous efforts to visualize the blood supply of the heart in living man, these studies have been largely of academic interest.

With presently developing attempts at direct surgical attack on lesions of the coronary arteries and the application of surgical procedures designed to effect revascularization of areas of the myocardium, effective roentgen methods for visualization of the coronary artery system become an increasingly important need.

It is the purpose of this presentation to discuss the problems of coronary arterial visualization and to describe the coronary arterial opacifications we have obtained in our employment of several procedures.

HISTORICAL

Roentgenologic demonstration of the coronary arteries in intact experimental animals was accomplished many years ago by Rousthöi.[12]

It was incident to the introduction of catheter thoracic aortography that the coronary arteries were successfully visualized in man. Jönsson[8] reported such visualization in 1948. Helmsworth, McGuire and Felson[6] also described their experiences in coronary arterial opacifications, utilizing procedures of catheter introduction into the ascending aorta.

In 1952, DiGuglielmo and Guttadauro[1] published an excellent treatise, "A roentgenologic study of the coronary arteries in the living," and presented in their publication diagrams of the roentgenologic appearance of contrast-filled coronary arteries. Copies of these very useful diagrams

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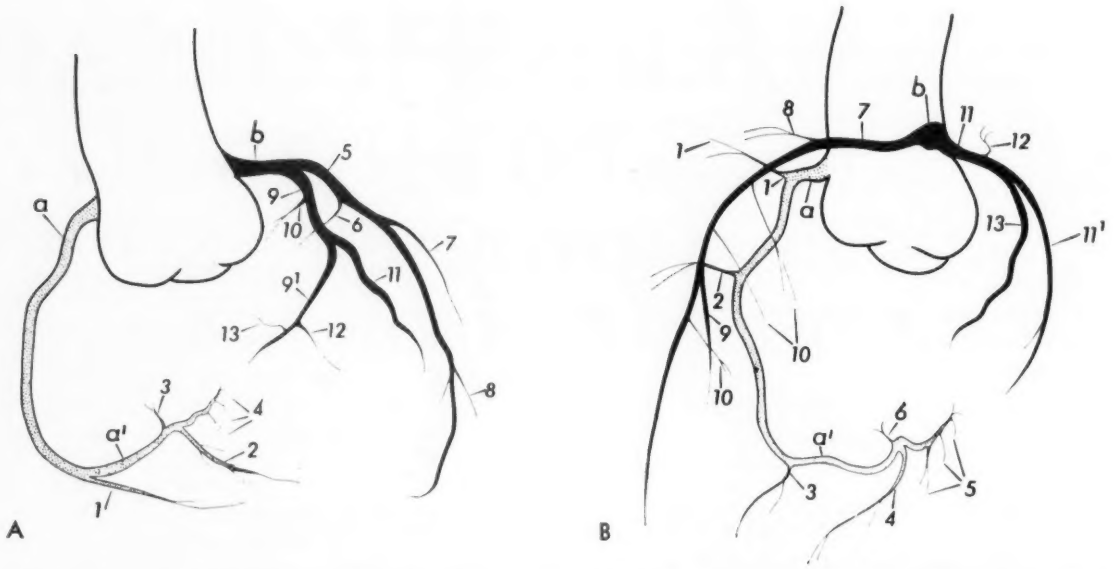

FIG. 1. Diagram of roentgenographic appearance of the coronary arteries filled with contrast agent. (A) Anteroposterior view. (a) *Right coronary artery* with (a^1) its second portion: (1) branch to the acute margin; (2) posterior interventricular branch; (3) posterior branch to the right atrium; (4) posterior branches to the right ventricle. (b) *Left coronary artery:* (5) anterior interventricular branch with (7 and 8) left branches; (6) left conus branch; (9) circumflex branch with (9^1) its second portion; (10) anterior branch to the left atrium; (11) branch to the obtuse margin; (12) posterior branch to the left ventricle; (13) posterior branch to the left atrium. (B) Lateral view. (a) *Right coronary artery* with (a^1) its second portion: (1) right conus branch; (2) anterior branch to the right ventricle; (3) branch to the acute margin; (4) posterior interventricular branch; (5) posterior branches to the left ventricle; (6) posterior branch to the right atrium. (b) *Left coronary artery:* (7) anterior interventricular branch with (9) the first left branch; (8) left conus branch; (10) septal branches; (11) circumflex branch with (11^1) its second portion; (12) anterior branch to the left atrium; (13) branch to the obtuse margin. (Reproduced with permission of Di Guglielmo, L., and Guttadauro, M., and *Acta radiologica.*)

are shown in Figure 1. These authors reported on the coronary visualizations obtained in 159 thoracic aortographies (146 examinations by catheter aortography and 13 by introduction of a cannula through the carotid artery). Simultaneous biplane filming was employed. Of the 159 examinations, coronary visualization resulted in 112, with failure of coronary opacification in 47. The important reasons for the failures are described in their report. These authors also reported that they were unable to visualize the coronary arteries by intravenous angiocardiography in a series of 48 studies. However, they were able to obtain some opacification of certain portions of the coronary arterial system in 9 of 121 selective catheter angiocardiographies.

Observations regarding angiocardiographic demonstration of coronary arteries were reported by Gordon, Brahms and Sussman,[5] with partial demonstration of coronary arteries in but 10 of 1,200 peripheral venous angiocardiographies.

Again, in 1954, DiGuglielmo and Guttadauro[2] reported their observations regarding anatomic variations of the coronary arteries when opacified by thoracic aortography.

More recently, Thal, Lester, Richards and Murray[14] published an article on coronary arteriography in arteriosclerotic heart disease. They employed a No. 10 Lehman thin-wall catheter inserted through the right brachial artery and positioned with its tip 2 inches above the sinus of Valsalva. These authors reported on experiences with 18 coronary arteriographies, detailing 5 cases in which coronary artery disease was clinically suspected.

Direct introduction of opaque medium into the aorta by transthoracic needle puncture of the aorta with subsequent coronary arterial opacification was described by Radner[11] in 1945. The procedure involves the insertion of a needle through the sternum into the ascending aorta. In the 5 performances reported there were 2 complications. Only faint opacification of the proximal portions of the coronary arteries is described in the publication.

Hoyos and del Campo[7] in 1948 obtained coronary artery opacification by parasternal needle puncture of the ascending aorta.

Wickbom[15] in a report of a fatality resulting from his technique of "thoracic aortography after direct puncture of the aorta from the jugulum" mentions coronary artery opacification as occurring in this case.

Dotter and Lukas[4] devised a double-lumen catheter, incorporating an inflatable rubber balloon connected to one lumen of the catheter. Dotter[3] has employed such a catheter in the ascending aorta, momentarily occluding the ascending aorta with simultaneous injection of radiopaque medium through the other lumen of the catheter. With this aortic occlusive technique, he has obtained excellent coronary arterial opacifications in experimental animals (dogs).

METHODS

Interest in coronary arterial opacification in the Department of Radiology at our institution originated with our employment of catheter thoracic aortography in 1948. In the performance of such aortography, we occasionally achieved excellent coronary arterial opacification. However, dissatisfaction with the opaque catheters then available prompted one of us (J.S.L.) to devise a so-called aortography catheter* to effect improvement in aortography and coronary artery opacification.

Coincident with our adoption of cardiac ventriculography[9] (transthoracic needle puncture opacification of the left—or right—ventricle) and the not infrequent visualization of the coronary arteries effected by this procedure, our interest in coronary arteriography was given further stimulus.

Recently, in an attempt to improve our thoracic aortographies and coronary artery opacifications, we have employed a procedure of intrathoracic needle puncture of the ascending aorta, utilizing a suprasternal approach and advancing the needle tip to a position slightly above the aortic sinuses, with rapid injection of radiopaque medium. When adapted to thoracic aortography, the procedure is termed "suprasternal thoracic aortography;" when applied primarily to coronary artery visualization, the name "suprasternal transaortic coronary arteriography" identifies the procedure. Communications regarding these procedures are in process of preparation for publication.

To improve our coronary arteriographies, in more recent studies we have induced cardiac arrest pharmacologically in conjunction with catheter thoracic aortography and with our suprasternal aortic needle puncture opacification techniques. In these studies we have used acetylcholine as the pharmacologic agent. Our experience with this approach toward improvement of coronary visualization has been most interesting and has indicated to us that some improvement in coronary arterial opacification can be accomplished on occasion. Complete details of our experiences with cardiac arrest coronary arteriography in both animals and humans are the subject of another paper now in preparation for publication.

In Tables I, II, III and IV are shown the coronary arterial and aortic opacifications resulting from each of the several procedures we have employed.

Analysis of each procedure was made on the following basis: Patients under fourteen years of age were not included for the reason that our series of cardiac ventriculographies was composed entirely of pa-

* Manufactured by United States Catheter Company, Glens Falls, New York.

TABLE I

CATHETER THORACIC AORTOGRAPHY

Attempted Examinations		44
Number of Patients	44	
Age Range	14 to 64 years	
Failures (inability to place catheter tip in ascending aorta)		4

Purpose of Study	Number of Examinations
Evaluation for aortic coarctation	20
Evaluation for aortic insufficiency	9
Evaluation for patent ductus arteriosus	7
Evaluation for aortic-pulmonary septal defect	5
Evaluation of superior mediastinal mass	2
Evaluation for aneurysm of innominate artery	1

Opacification Grading (40 Examinations)	Ascending Aorta	Coronary Arteries	
		Right	Left
Excellent	14 (35%)	7 (18%)	5 (12%)
Good	6 (15%)	5 (12%)	3 (8%)
Fair	8 (20%)	7 (18%)	7 (18%)
Poor	12 (30%)	9 (22%)	10 (25%)
No Visualization	—	12 (30%)	15 (37%)

Excellent visualization—both coronaries	4 (10%)
No visualization—either coronary	10 (25%)

tients fourteen years of age or older. This holds true also for our suprasternal needle opacification studies. Moreover, it has been our repeated observation that catheter thoracic aortographic techniques result in better coronary arterial system visualization in infants and young children than in adolescents and adults. The majority of our catheter thoracic aortographies have been performed in infants and young children. Furthermore, we feel that coronary arteriography has a more practical application to adults than to children.

We have also excluded all examinations in which the study was performed by single filming rather than by serial filming technique. For this reason, a number of our early catheter thoracic aortography cases have been eliminated from this analysis.

All examinations in each of the several procedures have been made in *single* plane only. Unquestionably, simultaneous biplane studies would have been advantageous; unfortunately, however, biplane equipment has not been available to us.

The majority of our catheter thoracic aortographies and cardiac ventriculographies have been performed in lateral projection with the patient recumbent. The suprasternal needle opacification studies

TABLE II

CARDIAC VENTRICULOGRAPHY

Attempted Examinations		230
Number of Patients	199	
Age Range	16 to 64 years	
Failures		28
Unintentional injection right ventricle	12	
Ventricle not penetrated	7	
Intramyocardial injection	5	
Needle tip too high	2	
Equipment failure	2	

Purpose of Study	Number of Examinations
Quantitation of mitral insufficiency	127
Evaluation for any mitral insufficiency	43
Evaluation of surgical correction of mitral insufficiency	15
Evaluation for any left ventricular aneurysm	14
Evaluation for congenital heart disease (principally interventricular septal defect)	11
Evaluation of aortic valve	10
Evaluation for aortic aneurysm	7
Evaluation of surgical correction of left ventricular aneurysm	2
Evaluation for aortic coarctation	1

Opacification Grading (202 Examinations)	Ascending Aorta	Coronary Arteries	
		Right	Left
Excellent	105 (52%)	46 (23%)	13 (7%)
Good	47 (23%)	60 (29%)	22 (11%)
Fair	38 (19%)	36 (18%)	33 (16%)
Poor	12 (6%)	36 (18%)	93 (46%)
No Visualization	—	24 (12%)	41 (20%)

Excellent visualization—both coronaries	13 (7%)
No visualization—either coronary	10 (5%)

have all been performed in supine, anteroposterior projection. Possible discrepancies in comparison of the coronary visualizations resulting from each procedure with regard to the projection employed will be pursued in our Discussion.

The opaque medium used in our earlier catheter thoracic aortographies and cardiac ventriculographies was a brand of iodopyracet (diodrast†) 70 per cent. In the more recent catheter thoracic aortography and cardiac ventriculography procedures and in all of our suprasternal needle opacification studies, we have used a diatrizoate methylglucamine radiopaque (hypaque† or cardiografin‡) in either 85 or 90 per cent concentration. The amount of radiopaque medium injected has varied with the weight and the size of patient from 20 cc. to 45 cc.

TABLE III

SUPRASTERNAL THORACIC AORTOGRAPHY

Attempted Examinations		107
Number of Patients	90	
Age Range	15 to 60 years	
Failures		30
Unsuccessful satisfactory puncture	25	
Equipment failure	3	
Inadvertent pulmonary artery injection	2	

Purpose of Study	Number of Examinations
Quantitation of aortic insufficiency	66
Evaluation of coronary artery system	38
Evaluation for patent ductus arteriosus	3

Opacification Grading (77 Examinations)	Ascending Aorta	Coronary Arteries	
		Right	Left
Excellent	46 (60%)	13 (17%)	12 (15%)
Good	11 (14%)	12 (15%)	14 (19%)
Fair	13 (17%)	10 (13%)	23 (30%)
Poor	7 (9%)	13 (17%)	16 (21%)
No Visualization	—	29 (38%)	12 (15%)

Excellent visualization—both coronaries	9 (12%)
No visualization—either coronary	9 (12%)

† Winthrop Laboratories, New York, New York.
‡ E. R. Squibb and Sons, New Brunswick, New Jersey.

TABLE IV

CARDIAC ARREST CORONARY ARTERIOGRAPHY

(Acetylcholine-Induced)

Attempted Examinations		22
Number of Patients	22	
Age Range	25 to 60 years	
Failures (para-aortic injection)		1

Purpose of Study	Number of Examinations
Evaluation of Coronary Artery System	22

Opacification Grading (21 Examinations)	Ascending Aorta	Coronary Arteries	
		Right	Left
Excellent	21 (100%)	6 (29%)	10 (48%)
Good	—	2 (9%)	4 (19%)
Fair	—	1 (5%)	4 (19%)
Poor	—	7 (33%)	2 (9%)
No Visualization	—	5 (24%)	1 (5%)

Excellent visualization—both coronaries	5 (24%)
No visualization—either coronary	0

In our early catheter thoracic aortographies, injection was performed by manual syringe pressure. In the later portion of the studies and in all of our cardiac ventriculographies and suprasternal needle aortographies, a mechanical pressure injector has been utilized.

Injection rate for all patients and examinations has varied from 1.5 to 3.5 seconds, the majority having been made in approximately 2.0 seconds.

RESULTS

In grading each procedure from the standpoint of the quality of coronary arterial visualization achieved, we have set forth the number of attempted procedures, the number of patients and their age ranges, and the primary purposes for which the procedures were performed. The proportion of failures to complete the procedure and the reasons for such failures are also tabulated. Finally, we have evaluated for each of the several procedures the

degree of coronary visualization or lack of visualization resulting from each examination. We considered it advisable to appraise each (the right and the left) coronary artery separately.

In assessing the *right* coronary artery, we have considered the course of its main trunk and have applied to it the classification of "excellent," "good," "fair," "poor," or "no visualization." These terms have been applied to the degree and clarity with which the main trunk throughout its major course was demonstrated. While we are, of course, concerned with the branch vessels arising from the main trunk, we consider them sufficiently variable anatomically to exclude them from exact tabulation.

These same criteria were applied to the *left* coronary artery, except that the appraisal was made on the basis of visualization of the short main trunk and of *both* its anterior descending and circumflex branches. If only one of these major branches (anterior descending or circumflex) was visualized, we relegated the left coronary artery classification to the "poor" category, regardless of the degree of opacification of the visualized branch.

The opacification of the entire ascending aorta has been classified on a similar basis of "excellent," "good," "fair," "poor," or "no visualization."

In an attempt to obtain uniform and consistent evaluations in what might be considered a difficult classification estimation, all of the studies for all procedures have been evaluated by one of us (J.S.L.).

DISCUSSION

Catheter Thoracic Aortography. The group of catheter thoracic aortographies we have studied is admittedly small in number. Since we have purposefully excluded all examinations in patients under fourteen years of age in order to analyze a series comprising an age group comparable to those of our other procedures, the majority of our catheter thoracic aortographies have been omitted from this analysis. The 44 examinations analyzed were made in one projection only (as were other procedures described in this communication), usually the lateral projection.

Of the 9 examinations in which the primary purpose of the procedure was evaluation for aortic valvular insufficiency, 5 exhibited some regurgitation of opaque medium into the left ventricle. Such regurgitant opacification of the left ventricular chamber may obscure the outlines of the coronary vessels, particularly the anterior descending branch of the left coronary artery. In the presence of patent ductus arteriosus or aortic-pulmonary septal defect, the hemodynamics of the aorta may be considerably altered and pulmonary vascular opacification usually occurs in such cases. These factors may seriously interfere with coronary arterial visualization.

In the 44 attempted catheter thoracic aortographies which were analyzed, there was one fatality resulting from the procedure. The aorta and innominate artery in this patient were greatly distorted by a superior mediastinal neoplastic mass, and the catheter tip was inadvertently placed in the distorted innominate artery. Practically the entire dose of radiopaque substance entered the right carotid artery; hemiplegia developed, and the patient expired three days following the attempted aortography.

Of the 40 examinations in which the catheter was successfully positioned in the ascending aorta, its tip was in the proximal third in 25; in the middle third in 12; in the upper third in 2, and in one instance the catheter tip was in the left ventricle. Our most satisfactory coronary arterial opacifications have been observed when the catheter tip was in the lower third of the ascending aorta. The majority of our catheter aortographies in this analysis were performed in the early days of our experience, at a time when we had only open-end aortography catheters available. Placement of the tip of any open-end catheter in one of the aortic sinuses may result in ineffective and incomplete coronary arterial visualization. We are now using a closed-

end, multiple side hole catheter, with all openings located in the distal 16 mm. of the catheter. The catheter tip is positioned approximately 1.0 cm. above the level of the aortic sinuses. We believe that this technique, with employment of some type of pressure injector to insure an approximate 2-second injection rate, results in improved coronary arteriography by catheter thoracic aortography. An illustration of successful opacification of *both* coronary arteries by catheter thoracic aortography is shown in Figure 2. A multiple side hole catheter was used, and there is regurgitant opacification of the left ventricle because of rheumatic aortic valvular insufficiency.

Figure 3 illustrates opacification of the *right* coronary artery only. In this case, the tip of an end hole catheter had been inadvertently placed in the right aortic sinus and practically the entire injected amount of radiopaque medium entered and opacified the right coronary artery. There were no sequelae incident to this injection.

FIG. 2. Catheter thoracic aortogram. Opacification of both coronary arteries. Note deformity of aortic valve and regurgitant opacification of the left ventricle. Rheumatic aortic valvular insufficiency.

FIG. 3. Catheter thoracic aortogram. Opacification of the right coronary artery. Tip of an end-hole catheter inadvertently placed in right aortic sinus. No sequelae. No significant electrocardiographic changes.

The procedure of catheter thoracic aortography for coronary arteriography involves the introduction of a catheter into a peripheral artery and its passage into the ascending aorta. Such an introduction necessitates a cut-down exposure of the artery and an incisional opening of the arterial wall. With withdrawal of the catheter, either ligation of the artery or repair of the arterial incision is necessary. Undesirable sequelae may result from such cut-down and repair, since attempted repair may effect actual closure of the arterial lumen, compromising the arterial vascular supply to the limb. At present, we are exploring the advantages of the Seldinger[13] technique of percutaneous introduction of a plastic catheter into a radial or femoral artery, and are using an Odman[10] radiopaque plastic catheter.

Cardiac Ventriculography. The procedure of cardiac ventriculography has yielded some satisfactory coronary opacifications, as illustrated in Figures 4 and 5.

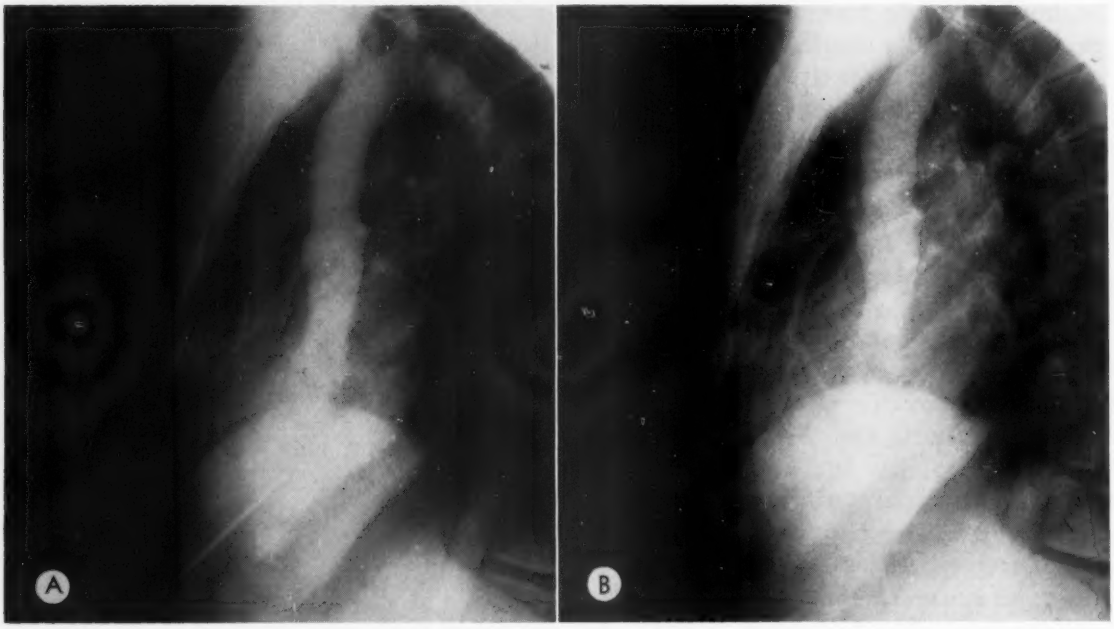

FIG. 4. Cardiac ventriculograms. (*A*) Completion of injection of 35 cc. 90 per cent opaque medium. Opacification of both coronary arteries. (*B*) Two seconds after completion of injection. Emptying of the left ventricle permits visualization of branches of the coronary arteries.

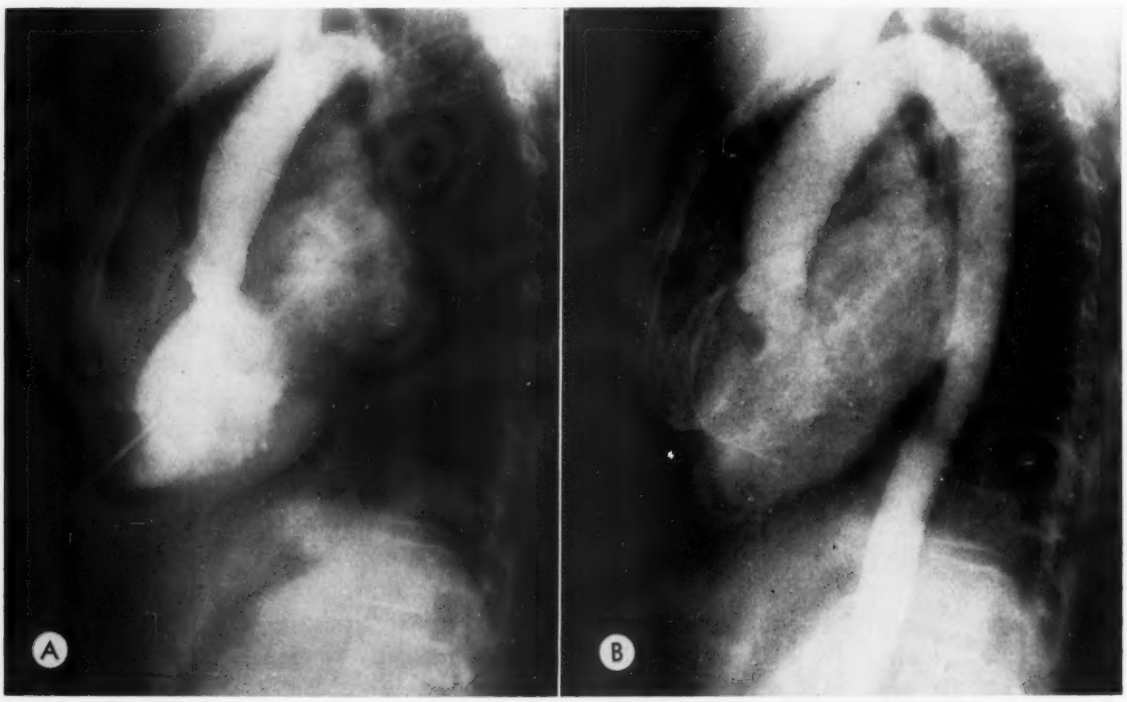

FIG. 5. Cardiac ventriculograms. (*A*) Completion of injection of 38 cc. 85 per cent opaque medium. Note slight regurgitant opacification of the left atrium. Rheumatic mitral valvular insufficiency. (*B*) One second after completion of injection. Emptying of the left ventricle permits visualization of the coronary arteries. Note thickening and slight deformity of the aortic valve cusps. Rheumatic aortic valvular disease.

Of 230 attempted cardiac ventriculographies—left, we failed to effect satisfactory study in 28, for the reasons outlined in Table II. The majority of the studies were designed for quantitation or evaluation of mitral insufficiency; in practically all of these there was some degree of regurgitant opacification of the left atrium, indicating the presence of dynamic mitral valvular insufficiency. In a considerable number, the enlarged left atrium, opacified with radiopaque substance, obscured to some extent the circumflex branch of the left coronary artery, as shown in Figure 6. In these cases, the opacification of the *left* coronary artery was classified as "poor," even though the anterior descending branch was well demonstrated.

When cardiac ventriculography is employed in patients with marked cardiomegalia and particularly when there is opacification of an enlarged left atrium and/or left ventricle, good demonstration of coronary arteries and their branches may not result. In aortic valvular disease, the altered hemodynamics are such that when the opacifying agent is injected directly into the left ventricular cavity, there may be an insufficient concentration of the opaque medium at the coronary ostia for satisfactory opacification of the coronary arteries.

FIG. 6. Cardiac ventriculogram. Opacification of both coronary arteries. Regurgitant opacification of the left atrium due to mitral valvular insufficiency partially obscures the circumflex branch of the left coronary artery.

Cardiac ventriculography is a procedure not devoid of hazard. Inadvertent injection of high-concentrate radiopaque medium into the myocardium is the gravest danger. In this series of 230 examinations, there have been 2 fatalities attributable to such intramyocardial injection.

Suprasternal Thoracic Aortography. The technique of needle puncture of the thoracic aorta, with positioning of the needle tip in the proximal third of the ascending aorta for rapid radiopaque injection, is a relatively new development. As previously mentioned, when this procedure is performed primarily for coronary arterial demonstration, we have elected to call it "suprasternal transaortic coronary arteriography." Details of a study of a group of patients having clinical evidence of coronary artery disease and upon whom this procedure was performed are to be reported elsewhere. In the present discussion, we have included the entire group of patients who had thoracic aortography studies by needle puncture opacification of the ascending aorta. Of 107 attempted examinations, we failed to obtain a successful study on 30 occasions for reasons listed in Table III.

In the majority of instances it was possible to place the needle tip at the desired location above the aortic sinuses and to deliver a sufficient quantity of high-concentrate radiopaque substance to effect excellent opacification of the aortic sinuses and the ascending aorta. Thus, a number of satisfactory coronary arterial visualizations were achieved, as shown in Figures 7, 8 and 9. It is to be noted that the greater number of these studies were performed for purposes of quantitation of aortic valvular

FIG. 7. Suprasternal thoracic aortogram. Completion of injection of 35 cc. 90 per cent opaque medium. Opacification of both coronary arteries and their major branches. Note regurgitant opacification of the left ventricle. Rheumatic aortic valvular insufficiency.

insufficiency in patients who had clinical evidence of aortic valvular disease.

It has been our frequent observation that with marked aortic valvular insufficiency, the considerable reflux of the injected radiopaque substance into the left ventricle, which occurs in such cases, may not allow a sufficient concentration of radiopaque material to enter the coronary arteries for their satisfactory opacification. It is, however, noteworthy that on occasion excellent coronary visualization may result even in the presence of a high degree of insufficiency of the aortic valve. Explanation for the above observations probably must await an opportunity for correlative study of surgical with necropsy findings regarding the status of the coronary arteries, and comparative interpretations of coronary arteriography.

The ascending aorta is usually dilated in dynamic aortic valvular disease, and such dilatation with its resultant increased radiopaque dilution is an additive factor in militating against the most satisfactory filling of the coronary arteries. In the absence of dynamic aortic valvular disease, we have achieved *some* degree of opacification of the coronary arteries rather regularly. In 38 of our 77 aortic opacification studies, the examination was performed primarily to evaluate the status of the coronary arteries. All of the 38 patients had clinical evidence of coronary artery disease. In such instances, the problem presents itself as to whether *non*visualization of a coronary vessel denotes a degree of occlusive disease or insufficient radiopaque contrast filling. This aspect will be discussed in a later portion of this paper.

All of the suprasternal thoracic aortographies were performed with the patient in supine position, anteroposterior projection, with no attempt to rotate the patient other than the head and neck. Such projection rather regularly places the right

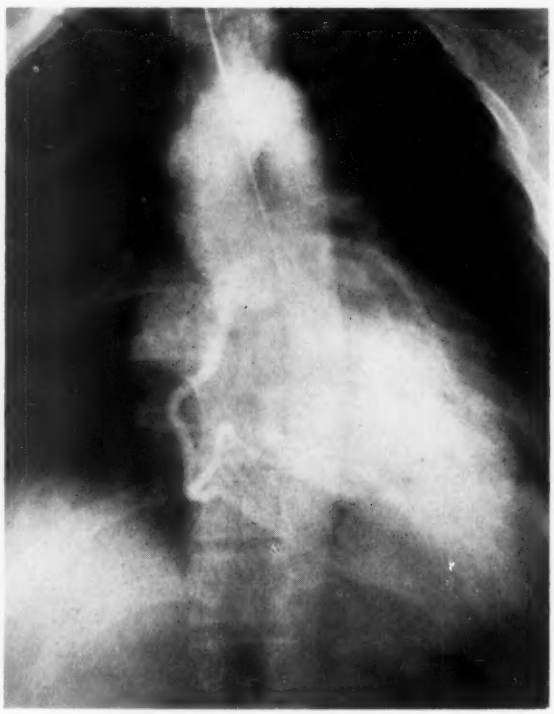

FIG. 8. Suprasternal thoracic aortogram. One second after completion of injection of 35 cc. 85 per cent opaque medium. Opacification of both coronary arteries. Note evidence of aortic valvular insufficiency.

coronary artery at a point overlying the somewhat dense structure of the thoracic spine; hence, demonstration of the right coronary artery is difficult. As with other procedures discussed in this presentation, simultaneous biplane projections would have been of value.

The presence of cardiomegalia with increased density of the heart again offers an appreciable problem in effecting the most satisfactory delineation of the coronary arteries and their branches.

The procedure of suprasternal thoracic aortography (and its component, suprasternal transaortic coronary arteriography) is relatively new, and we can find no reference in the literature to a similar technique, wherein the needle is advanced down the ascending aorta to place its tip near the aortic sinuses. The true and final value of the procedure as a means of effecting coronary arteriography is yet to be fully determined. It is not without hazard and, while we have experienced no fatalities,

Fig. 9. Suprasternal thoracic aortogram. Completion of injection of 40 cc. 90 per cent opaque medium. Opacification of both coronary arteries. Note minimal aortic valvular deformity and slight regurgitant opacification of the left ventricle.

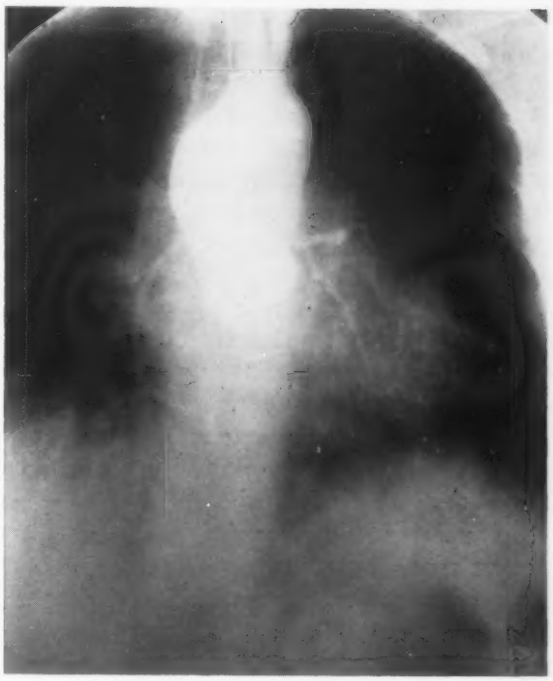

Fig. 10. Cardiac arrest coronary arteriogram. Cardiac arrest of four seconds' duration obtained with 5 mg. of acetylcholine. Injection of 35 cc. of 90 per cent opaque medium during the first two seconds of arrest. Opacification of both coronary arteries.

there have been instances of evidence on following roentgenographic study of bleeding within the superior mediastinum. None of these, however, required specified treatment or surgical intervention. A few instances of some hemorrhage within the pericardial sac have been observed. These particular complications and the risks of intramural aortic and para-aortic injection are being considered in detail in the previously mentioned paper in preparation.

Cardiac Arrest Coronary Arteriography (Acetylcholine-Induced Cardiac Arrest and Aortography). So far, our experience with pharmacologically-induced cardiac arrest and simultaneous intra-aortic injection of high-concentrate radiopaque contrast agents in human beings has suggested that improved coronary arterial opacification can be effected by this procedure. An example of coronary opacification obtained by cardiac arrest coronary arteriography is shown in Figure 10.

All of the studies analyzed in Table IV

were performed on patients with clinical evidence of coronary artery disease, in whom the existence of varying degrees of luminal narrowing and/or occlusive changes in the coronary arteries and their branches is to be anticipated. Consequently, interference with adequate radiopaque filling of these vessels may be expected. Such inadequate filling plus the direct demonstration of areas of narrowing and obstruction of the coronary arteries form the basis for coronary arteriographic diagnosis of coronary artery disease.

Cardiac arrest coronary arteriography may well become the method of "choice" for coronary arteriography if (1) it can be demonstrated conclusively that normally patent coronary arteries will fill regularly with high concentrations of radiopaque medium injected into the proximal third of the ascending aorta during a period of cardiac asystole; (2) such filling is more adequate than similar injection procedures unaccompanied by cardiac arrest; and (3) the dangers of such induced cardiac arrest are not prohibitive. While our evaluation of the usefulness and hazards of cardiac arrest coronary arteriography is not yet complete, we have been impressed with the very noticeable improvement in coronary visualizations that we have achieved in experimental animals (dogs). We are pursuing the study of various factors, such as types of pharmacologic agent, dose, rate and route of injection, time interval between injection of the arresting agent and radiopaque medium injection, and reactions and physiologic changes produced by cardiac arrest coronary arteriography.

In our 22 human patients on whom we have thus far performed this procedure, we have employed several dose levels of acetylcholine and have induced cardiac arrest for intervals varying from two to thirty-two seconds. However, in several patients, only a short period of bradycardia resulted. Of the 22 examinations, 19 were suprasternal needle puncture procedures and the other 3 were catheter thoracic aortographies. A short episode of pronounced convulsion in one patient resulted in displacement of the intra-aortic needle, and the entire quantity of radiopaque medium infiltrated the para-aortic tissues. This patient experienced shock and substernal pain, but recovered and was discharged from the hospital five days following the procedure. Other less severe convulsions have been noted on several occasions, and it is our present observation that acetylcholine-induced cardiac arrest presents some risks.

GENERAL CONSIDERATIONS

We believe that there is a genuine need for a technique of coronary arteriography which is efficient, reliable and without appreciable hazard. Roentgenologic demonstration of coronary arteries can be accomplished by several methods.

While cardiac ventriculography can effect coronary artery opacification with considerable frequency, this procedure has the disadvantage of dense opacification of the left ventricular cavity, which may obscure portions of the coronary artery system since the injected radiopaque substance is somewhat diluted when it reaches the coronary ostia. Cardiac ventriculography is not an unhazardous procedure, and we do not believe it has proved itself to be the "choice" technique for coronary arteriography.

Suprasternal needle puncture procedures (suprasternal thoracic aortography and suprasternal transaortic coronary arteriography) are likewise not without danger. The possibility of intramural aortic injection of radiopaque medium and of aortic bleeding must not be overlooked. In a percentage of our examinations, we have not been successful in penetrating the aorta by the needle. It is our conviction that there is need for further experience with such needle puncture procedures before their usefulness can be fully established.

In our opinion, catheter thoracic aortography appears to be the most efficacious method of delivering relatively undiluted,

high-concentrate radiopaque medium to the coronary ostia in a quantity sufficient to effect good coronary artery visualization. The ability to deposit the radiopaque substance at a predetermined level in the ascending aorta, with regularity, is of considerable advantage. The risks involved in this procedure are principally those related to malposition of the catheter tip and those incident to repair or closure of the artery through which the catheter is introduced.

Cardiac arrest coronary arteriography seems to merit further investigation. The potential hazards of inducing a period of cardiac asystole are as yet indeterminate. Thus far, our experience would suggest that this procedure may be of considerable worth in consummating satisfactory coronary artery visualization, but we are not unconcerned as to its possible dangers.

Momentary occlusion of the aorta can produce highly satisfactory coronary opacification in experimental animals. However, we are cognizant of the risks incident to the alteration of pressure within the left ventricle, in the proximal portion of the aorta, and in the coronary artery system which occur at the time of aortic occlusion. Likewise, we are not unmindful of the possible hazards associated with introduction and withdrawal of a double-lumen catheter with an attached distensible balloon.

The concept of combining induced cardiac arrest with simultaneous catheter balloon occlusion of the ascending aorta was suggested to us by Dotter,[3] and we have utilized this technique in coronary arteriography on experimental animals. Some very excellent coronary arteriograms, with demonstration of minute branches, have resulted. The possible application of this procedure to living man deserves thoughtful consideration since, at the time of cardiac arrest, the driving force of the left ventricular contractions is no longer functioning, and balloon occlusion of the ascending aorta does not precipitate hypertensive states in the coronary arteries, aorta and left ventricle. Simultaneous biplane rapid serial filming during coronary arteriography is to be preferred to single plane film study, as aforementioned.

Cinefluorography would appear to offer an added advantage, and with development of image intensifiers of a size sufficient to include the entire adult heart within the field, cinefluorography and particularly simultaneous biplane cinefluorography may become the most satisfactory means of recording coronary artery opacification. With experimental animals, it is possible to obtain an infinite number of projections by rotating the animal during coronary opacification and employing cinefluorography. A rotating cradle has been devised in our department, which attains a full 180 degrees of rotation. The cinefluorographic coronary arteriographies which we have performed on dogs have yielded a pseudo-three dimensional effect and permitted demonstration of all portions of the opacified coronary arterial system. The application of such rotational techniques to coronary arteriography in man is intriguing.

The development of radiopacifying agents completely free of toxic properties is much to be desired and may resolve an important problem in coronary arteriography.

The technical difficulties of roentgenologic demonstration of the coronary arterial system in thick-chested persons, particularly those with cardiomegalia, is ever present. Since the human coronary arterial system does not have an anatomically fixed pattern and since there are variations in size and distribution of each coronary artery and in the number and distribution of their branches, there will always remain the difficulty of interpreting coronary arteriograms. Furthermore, one must be ever cognizant of the fact that *non*opacification of a coronary artery may either be an indication of partial or complete occlusion or of sufficient narrowing of its proximal or distal portions to prevent its opacification, or the coronary ostium may not have been exposed to a sufficient amount and

FIG. 11. Suprasternal thoracic aortogram. One second after start of injection of 40 cc. 90 per cent opaque medium. *Right coronary:* poor opacification; *left coronary:* defects in the anterior descending branch and poor filling of the circumflex branch. Palpable sclerotic and calcific plaques in both coronary arteries found at subsequent surgery (Vineberg procedure).

FIG. 12. Suprasternal thoracic aortogram. Completion of injection of 40 cc. 90 per cent opaque medium. *Right coronary:* irregular narrowing; *left coronary:* extensive constrictive defects. Calcific plaques and thickening in both coronary arteries and major branches found at subsequent surgery (Vineberg procedure).

concentration of contrast medium to effect adequate vessel filling. As previously mentioned, the arteriographic diagnosis of coronary artery disease rests on the demonstration of irregularity of the opacified vessel lumen, abnormal narrowing or areas of constriction, abnormal tapering and/or absence of vessel filling, or incomplete filling.

Figures 11, 12 and 13 are illustrations of abnormal coronary arteriograms with evidence of coronary artery pathology. The legends of these illustrations describe briefly the visual and palpatory observations regarding the epicardial portions of the coronary arteries, as noted at subsequent cardiac surgical procedures.

When one takes into consideration the motion of the heart, the velocity of blood flow, the possible narrowness of the lumen of a normal coronary artery as well as the small amount of contrast substance it may actually contain, one can appreciate that dynamic coronary artery system visualization may present many difficulties. Moreover, one must not overlook the fact that the pattern of disease in the coronary arteries is variable, and in some instances there are undoubtedly extensive arterial changes which do not sufficiently narrow or obliterate the arterial lumen to permit arteriographic detection.

SUMMARY

Experiences with coronary artery visualization, employing catheter thoracic aortography, cardiac ventriculography, suprasternal thoracic aortography and its component suprasternal transaortic coronary arteriography, and cardiac arrest coronary arteriography are described.

Assessment of coronary artery opacifications accomplished by each of these procedures is tabulated.

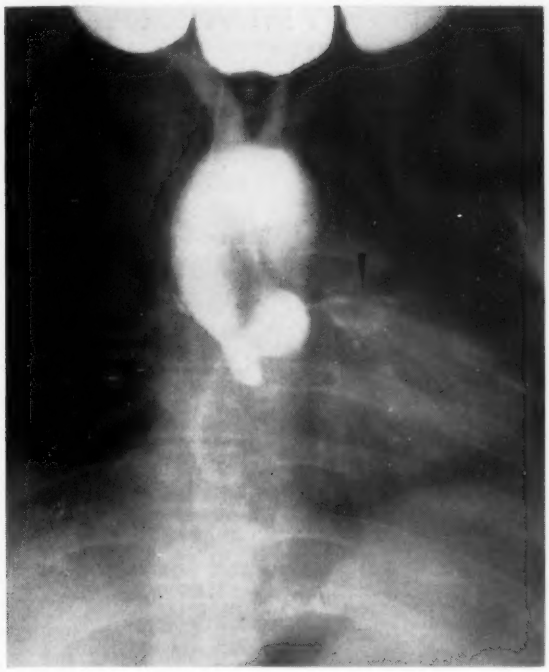

FIG. 13. Suprasternal thoracic aortogram. One second after start of injection of 35 cc. 90 per cent opaque medium. *Right coronary:* no evident lesion; *left coronary:* constrictive defect, proximal portion of anterior descending branch, and poor filling of both branches. Sclerotic, calcific changes in both branches of the left coronary artery found at subsequent surgery (Vineberg procedure).

The application of these several procedures to coronary arteriography is discussed.

A discussion of other techniques of coronary arteriography, including aortic occlusion coronary arteriography and cardiac arrest coronary arteriography plus aortic balloon occlusion, is presented.

A general consideration of various other aspects of coronary arteriography is included.

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A COMPARISON OF TECHNIQUES FOR VISUALIZATION OF THE CORONARY ARTERIES*

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AORTOGRAPHY and selective peripheral arteriography have made possible the visual differentiation between diffuse and segmental arterial disease and have thereby permitted the selection of candidates for direct arterial operations, such as by-pass or replacement grafting, or endarterectomy procedures. It is expected that the utilization of contrast material for the demonstration of the coronary circulation will: (1) enable the surgeon to differentiate between diffuse and segmental coronary occlusive disease; (2) permit him to choose a particular palliative procedure to increase myocardial blood flow; (3) provide an objective criterion for the evaluation of existing surgical procedures; and (4) aid in developing new operations to alleviate coronary insufficiency.

Until the advent of coronary arteriography, our impression of the relative incidence of segmental versus diffuse coronary occlusion was based on autopsy material from patients with far advanced terminal coronary disease, or from unselected autopsy material including incidental findings of asymptomatic coronary artery disease.[2] The demonstration of the coronary artery circulation at autopsy by means of dissection alone has its limitations in that only the relatively large branches may be studied. Injection of the coronary arterial tree with radiopaque material was carried out by Gross[5] in 1921. An improved method was introduced by Blumgart *et al.*[2] who used a colored radiopaque mass mixture and compared the roentgenographic findings with very careful dissections. Since the pioneer work of Grossman,[6] there has been a number of studies of coronary arteriography in dogs by various techniques.[3,9,11,12,13] It is now becoming feasible to visualize the coronary arterial tree roentgenographically by means of relatively safe procedures in patients with anginal symptoms, and even in those with advanced coronary occlusive disease.[4,7,8,10,14,15,16]

Unlike the extremity with obliterative arteriosclerotic disease, the heart is a highly irritable organ undergoing continuous rhythmic contractions. Arrhythmias, fibrillation, and arrest are potential hazards of coronary arteriography. It is of extreme importance, therefore, to evaluate the variables of technique to provide maximum safety from permanent or even temporary damage and yet achieve adequate coronary visualization. This study was designed to evaluate systematically various techniques for a safe and reliable method of coronary arteriography in dogs with myocardial infarcts secondary to ligation of the left anterior descending coronary artery.

MATERIALS AND METHODS

Comprehensive studies were done on a colony of 46 previously healthy mongrel dogs weighing between 10–20 kg. as follows: one week prior to ligation of the left anterior descending coronary artery, a baseline coronary arteriogram was taken, followed by a second post-ligation arteriogram in two to six weeks. The dog was sacrificed and autopsied forty-eight hours later.

A. Methods for Contrast Visualization. Comparisons were made of: (1) time and dosage factors in chemically induced cardiac arrest; (2) the filling of the arterial tree in the moving versus the arrested heart; (3) various radiopaque media in different concentrations; (4) the preferred location

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of the catheter tip; (5) hand injections versus automatic injector; (6) optimum pressure values for injection; (7) single table-top roentgenography versus the Schönander unit roentgenographic machine; and (8) exposure time of the subject during the various procedures.

The following method was found to be most satisfactory. The anesthetized dog (intravenous nembutal, 1.0 cc. per 5 kg.) was maintained on a slow intravenous drip of normal saline through a polyethylene catheter inserted in the jugular vein. The common carotid artery on the same side was isolated and occluded with a loop of silk suture after a No. 8 woven intra-arterial catheter was threaded retrograde to a distance of about 15 cm. Under fluoroscopic control the tip of the catheter was placed at the junction of the ascending aorta and the brachiocephalic artery. This was facilitated by introducing small bursts of 50 per cent hypaque and observing the pulse wave and direction of blood flow. The catheter was firmly secured to the syringe of the automatic injector (containing 25–50 cc. of radiopaque medium). The use of the special pressure apparatus enabled the operator to stand free of scatter radiation and permitted standardization of the technique of the examination. The dog was strapped in the right posterior oblique position. The technique employed was 100 ma., 0.1 second (10 mas.), and 75–80 kv. peak using an 8–1 grid.

A pre-injection baseline electrocardiogram was taken. While the tracing was observed, a bolus of 4–8 mg. of acetylcholine was injected rapidly into the vein and flushed through with normal saline. Three seconds after asystole was identified, the firing mechanism of the automatic injector was released (Fig. 1). When the plunger was within 5 cc. of completion, the exposure was made. Electrocardiographic monitoring was continued until the pre-injection baseline was obtained. The carotid arteriotomy was repaired with arterial silk.

B. Methods for Evaluating Myocardial Damages. Comparisons were made of: (1) electrocardiographic tracings taken before, during, and at periodic intervals after each procedure; (2) serum transaminase levels determined before and after each procedure; and (3) detailed histologic examinations of the heart carried out at autopsy with particular attention paid to any evidence of fresh damage (due to the last arteriography) superimposed on the old myocardial infarcts.

RESULTS

For optimum visualization 50 per cent hypaque proved to be superior to 90 per cent hypaque, 70 per cent urokon, 50 per cent cardiografin, 35 per cent and 70 per cent diodrast (Fig. 2). Thorotrast provided visualization equally as good as the hypaque. However, the use of thorotrast would be restricted particularly in younger patients to obviate the possible development of neoplasms of the reticuloendothelial system. Histologic studies have revealed thoratrast foreign body granulomata in the myocardium; such granulomata were not present when hypaque was used (Fig. 3). Higher concentrations of hypaque were found to cause a greater degree of electrocardiographic disturbances and yet not provide any better visualization of the coronary arteries. The automatic injector was found to be superior to hand injections and a pressure of 120 pounds per square inch over a four second period provided consistently adequate coronary visualization. The best location for the tip of the intra-arterial catheter was at the junction of the brachiocephalic artery and ascending aorta; when the tip of the catheter was moved down to the level of the sinuses of Valsalva, there was on occasion filling of one coronary artery only.

Acetylcholine arrest was the most significant addition to the procedure.[1] Four to 8 mg. of acetylcholine intravenously consistently produced a temporary asystole lasting approximately five to fifteen seconds. During this period of cardioplegia, the automatic injector was released, and the resultant visualization of the coronary

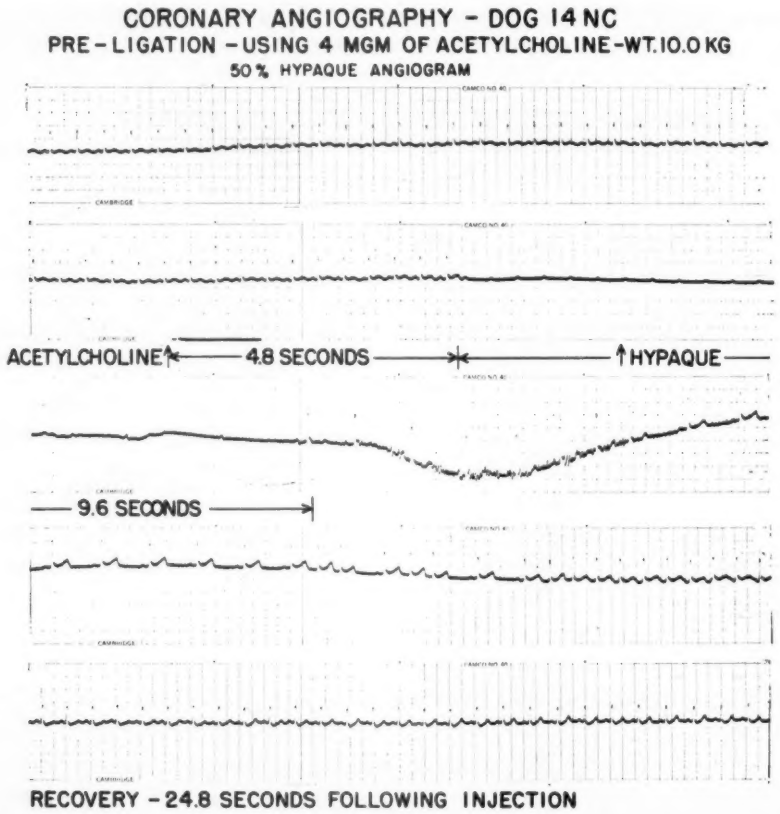

FIG. 1. A pre-ligation electrocardiogram with acetylcholine cardioplegia for 9.6 seconds. The radiopaque medium was injected three seconds after the onset of arrest. Full recovery occurred 24.8 seconds following the acetylcholine injection.

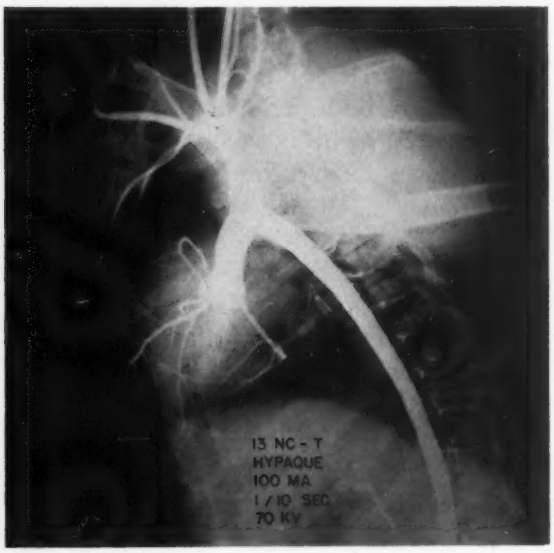

FIG. 2. A pre-ligation coronary arteriogram utilizing the single (T) exposure technique, 30 cc. of 50 per cent hypaque and acetylcholine cardiac arrest. (Reproduced by permission of the C. V. Mosby Company.)

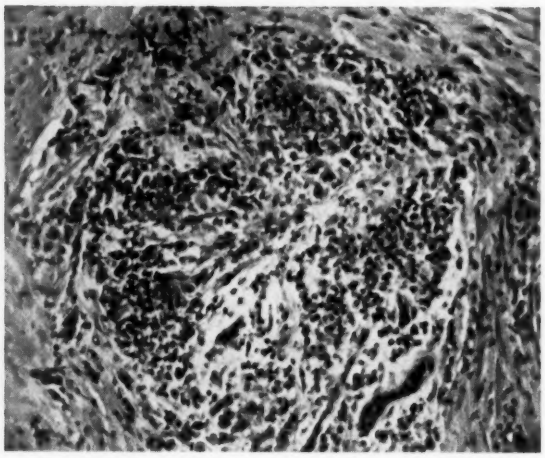

FIG. 3. A thorotrast granuloma in an old myocardial infarct near the point of ligation of a coronary artery following the use of thorotrast for coronary arteriography. (Masson; ×250.) (Reproduced by permission of the C. V. Mosby Company.)

vessels has been unexcelled in our experience (Fig. 2). The acetylcholine arrest did not cause residual damage even in the post-ligation dogs as judged by the electrocardiographic tracings, serum transaminase levels, and histologic studies. With or without acetylcholine arrest, coronary arteriography caused some temporary electrocardiographic changes (Fig. 4), with reversion to a normal pre-injection tracing within three to six minutes. In the control animals without arteriography, acetylcholine asystole also lasted between five to fifteen seconds with reversion to a normal pre-arrest tracing within three to five minutes (Fig. 5). There were no deaths attributable to the use of acetylcholine, and there was no difference in the effect of the drug between the pre-ligation and post-ligation groups (Table 1). The acetylcholine arrest also permitted the use of single exposure roentgenograms with a Potter-Bucky grid for greater detail, the exposure being coordinated with the onset of asystole as monitored by the electrocardiographic tracing. With experience, the single exposure roentgenogram proved to be superior to the Schönander multiple exposure technique. The coronary visualization is more adequate (Fig. 2 and 6) and the amount of radiation exposure to the personnel and the cost of equipment is markedly reduced. This technique thus brings coronary arteriography within the scope of departments of radiology or experimental surgery

Table I

CORONARY ARTERIOGRAPHY ACETYLCHOLINE INDUCED CARDIAC ARREST

	Pre-ligation (18)	Post-ligation (14)	All (32)
	sec.	sec.	sec.
Injection to Asystole	3.56	4.73	4.07
Asystole to Ventricular Contraction	8.59	8.26	8.48
Recovery	36.40	36.78	36.28

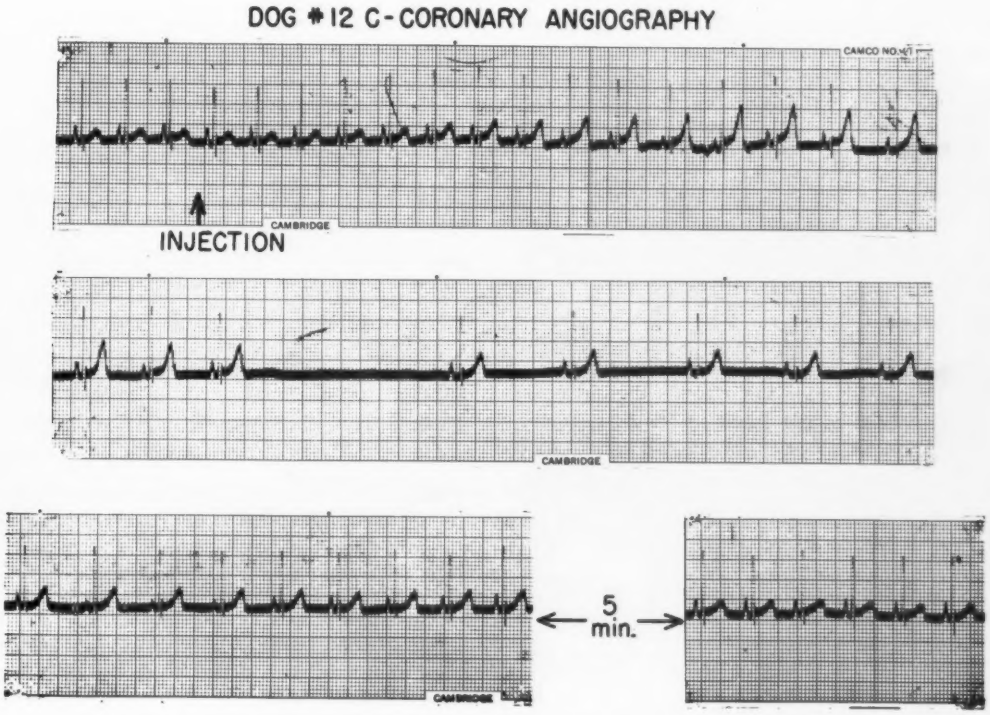

Fig. 4. A pre-ligation electrocardiogram showing transient S-T wave changes following the injection of 30 cc. of 50 per cent hypaque without acetylcholine arrest. Complete recovery occurred within five minutes.

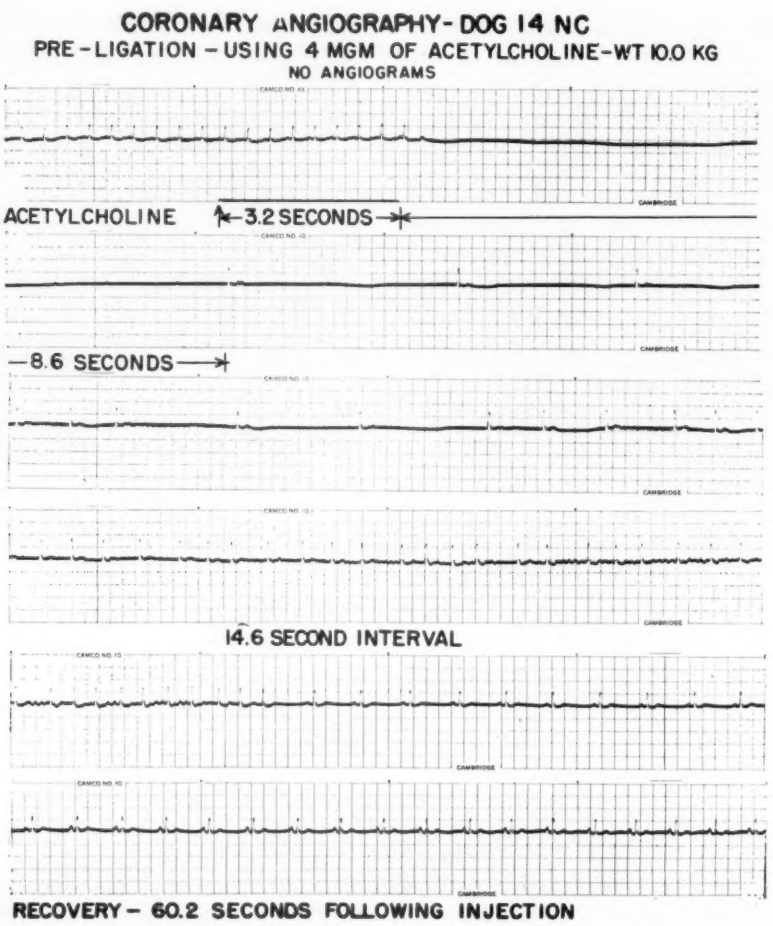

FIG. 5. A pre-ligation electrocardiogram with acetylcholine cardioplegia for 8.6 seconds; full recovery occurred 60.2 seconds following injection.

not equipped with a Schönander or similar unit.

Whereas ligation of the left anterior descending coronary artery produced a significant elevation in the serum transaminase levels, no change in the latter was noted following coronary arteriography, pre- or post-ligation and with or without acetylcholine arrest. Histologic studies of the hearts of the dogs sacrificed forty-eight hours after post-ligation coronary arteriography failed to reveal any evidence of acute morphologic changes superimposed on the old myocardial infarcts (Fig. 7).

COMMENT

In the dog with induced coronary arterial occlusion, coronary arteriography proved a safe procedure even with the use of temporary acetylcholine cardiac arrest. The latter permitted excellent visualization of the coronary arteries by employing the simplest roentgenographic equipment. This is in contrast to the method described by Miller *et al.*[12] who used potassium arrest and a pump-oxygenator resulting in the death of 5 out of 10 dogs.

The techniques of coronary arteriography described above not only permitted the detection of lesions created artificially (Fig. 8) but also aided in the visualization of the development of collateral arteries to ischemic areas (Fig. 9).

These safe yet effective techniques of coronary visualization should permit the use of coronary arteriography in the human patient with minimum risk. It is hoped that by the widespread practice of

Fig. 6. A pre-ligation coronary arteriogram utilizing the Schönander (S) multiple exposure technique, 30 cc. of 50 per cent hypaque and acetylcholine cardiac arrest. A significant loss of detail is noted as compared to the technique used in Figure 2. (Reproduced by permission of the C V. Mosby Company.)

coronary arteriography in patients with clinical coronary arterial insufficiency, a true perspective of the pathologic anatomy will be derived. This information may then be used to select, modify, and develop surgical procedures to alleviate coronary arterial insufficiency. It is felt that arterial occlusion in the larger coronary vessels will be more frequently treated by direct operative means; for this reason the localization of these occlusions by arteriography will gain increasing significance. The selection of candidates for direct coronary artery surgery versus the use of indirect palliative methods of increasing myocardial blood flow would be based on the interpretation of such arteriographic studies as advocated by Thal *et al.*[17]

SUMMARY AND CONCLUSION

1. Various techniques of coronary arteriography were compared in a colony of 46 mongrel dogs.

2. The injection of 30 cc. of 50 per cent hypaque with an automatic injector through an intra-arterial catheter with the tip located at the junction of the brachiocephalic artery and the ascending aorta consistently produced adequate visualization of the coronary arterial tree.

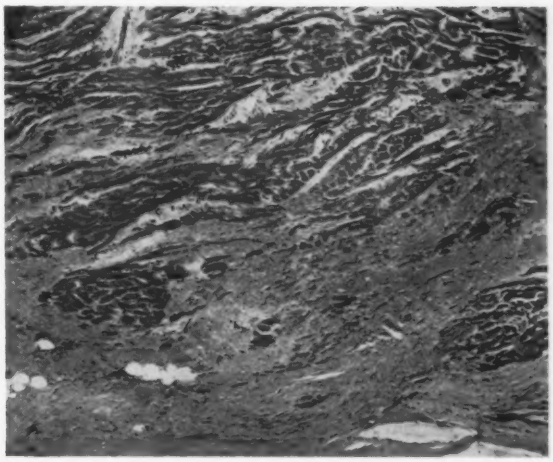

Fig. 7. A myocardial infarct in a dog four weeks following coronary artery ligation. No toxic changes induced by recent arteriography or intravenous acetylcholine are noted. (Masson; ×35.) (Reproduced by permission of the C. V. Mosby Company.)

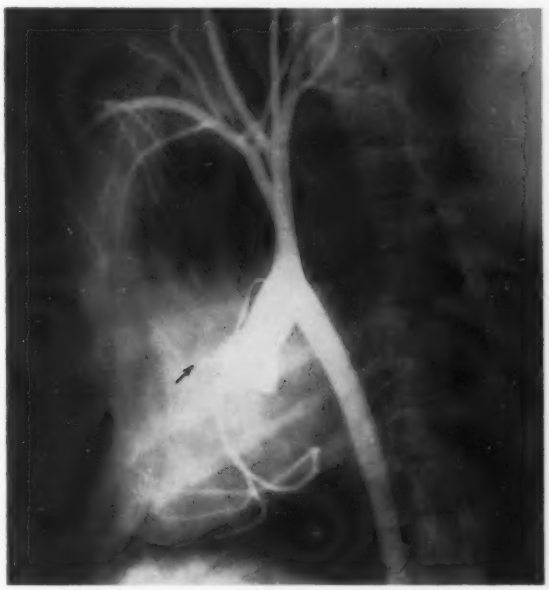

Fig. 8. A post-ligation coronary arteriogram indicating the point of ligation of the left anterior descending coronary artery (arrow). (Reproduced by permission of the C. V. Mosby Company.)

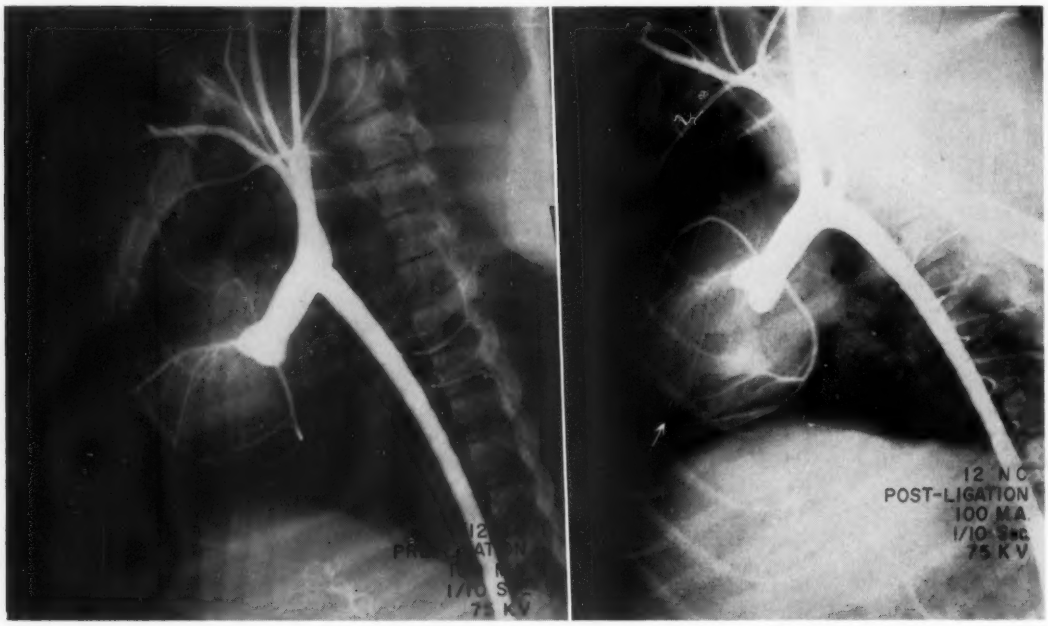

FIG. 9. Pre- and post-ligation coronary arteriograms, the latter four weeks following ligation of the left anterior descending coronary artery. The ligated artery is filled retrograde through a collateral channel (arrow) from the posterior descending branch.

3. The technique was improved by the use of temporary acetylcholine cardiac arrest. Acetylcholine cardioplegia provided a safe, transient asystole; reversion to normal occurred within two minutes.

4. Electrocardiographic monitoring, serum transaminase determinations, and histologic examinations detected no significant alterations following induced arrest and arteriography. Because of its alleged capacity for secondary damaging effects, the use of thorotrast should be restricted.

5. There apparently was no added hazard to the use of coronary arteriography and acetylcholine arrest in dogs with myocardial infarcts. Such experimentally induced lesions were depicted clearly.

6. It is proposed that coronary arteriography may be employed as a special diagnostic procedure in the evaluation of patients with coronary occlusive disease. The selection of candidates for direct coronary artery surgery versus indirect methods of increasing myocardial blood flow may be based on such arteriographic studies.

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CALCIFICATION OF THE CORONARY ARTERIES

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AT PRESENT, the clinical diagnosis of coronary atherosclerosis is made indirectly. When manifestations of ischemic heart disease are recognized, underlying coronary atherosclerosis is assumed to be present. If some independent technique could be used to assess coronary atherosclerosis, it would be of value to differentiate these two conditions and evaluate them separately. It is possible to estimate the luminal diameter of the coronary arteries in living man by coronary arteriography. However, this technique is not well suited to routine use and its application is limited because of the hazard involved. Radiopaque deposits are known to occur spontaneously in the coronary arteries. Roentgenographic examination for these should entail no risk to the patient and might be made routine, if useful information could be obtained.

Radiopaque densities in the coronary arteries consist principally of calcium. They rarely occur outside of atheromatous plaques after infancy. In 34 hearts (16 with myocardial infarction) removed at autopsy, the degree of stenosis in the coronary arteries, as judged following lipiodol injection, was found to parallel the occurrence of calcific coronary deposits.[7] Coronary calcifications have been seen during cardiac fluoroscopy, although not frequently.[2,4,6,8] One search in living man, employing spot roentgenograms, demonstrated their presence in 3 per cent of patients over forty years of age.[1] One autopsy study revealed them in 38 of 72 consecutive hearts.[3] The disparity in reported incidence at autopsy and that found during life suggests that the majority go unrecognized in life. To determine if better means might be found to search for these calcifications in life, we have performed a study of their size, location, and appearance in postmortem material.

METHODS AND MATERIAL

Roentgenographic Technique. After excision from the cadaver, the heart was roentgenographed before being examined further. The pulmonary veins and great veins had been cut at their entrance to the auricles. The right and left pulmonary arteries had been sectioned at or just beyond the bifurcation, and the aorta just distal to its valve. The heart was placed directly on film and roentgenographed, employing a self-rectifying roentgen-ray tube with a 1.4 mm. focal spot at 40 inch film distance. The first 56 hearts were positioned in the postero-anterior position (right ventricular wall and anterior septum nearest the film). The remaining hearts were in the anteroposterior position. With the tube at 40 inches, this change does not measurably alter the size of the shadows cast by calcification in the coronary arteries. After being roentgenographed, the hearts were cut, examined, and then stored at deep-freeze temperature until completion of the study. When examination of a film left doubt as to the location of a calcific deposit, the heart was unfrozen, examined, and re-roentgenographed after appropriate dissection.

Two types of roentgenographic film were used, each with a different exposure factor. The first 56 hearts were roentgenographed on Kodak Blue Brand screen film, contained in cardboard holders and exposed with 55 kv., 35 ma. sec. The remaining hearts were roentgenographed on Ansco Monopack, and exposed with 55 kv., 15 ma. sec. Both types of film were developed five minutes at 68° F. Best results were obtained with Ansco Monopack which is the most convenient.

Case Material. The 76 patients studied were autopsied at the Los Angeles County General Hospital. Forty-four were selected at random (but not by a planned scheme to randomize the case material) from the

Table I

AGE DISTRIBUTION BY DECADE AND SEX OF 76 CASES STUDIED

Age (yr.)	No. of Cases	
	Male	Female
40 and under	3	2
41–50	4	2
51–60	9	4
61–70	10	8
71–80	13	5
81–90	6	8
91–100	2	0
Totals	47	29

autopsy population over one year of age. To this number, 32 consecutive cases over one year of age were added.

Table 1 shows the distribution of ages by decade and sex of these patients.

RESULTS

Incidence of Calcifications. Sixty-one of 76 hearts (80 per cent) showed calcific lesions of the coronary arteries. Thirty-one of these also had calcification at another site, and in 7 extracoronary calcification predominated. Fourteen hearts of the 76 (17 per cent) showed no calcific lesion, and 2 of 76 (3 per cent) showed only valvular calcification.

When the incidence of calcification at various sites is considered in relation to age (Table II), it can be seen that before forty years of age, the majority of hearts showed no calcification. After forty-one years of age, the majority of hearts exhibited calcification. At all ages, calcified coronary arteries were the most common calcific lesion. The predominance of coronary calcification was particularly marked between the ages of forty-one and sixty and appeared to precede any large incidence of aortic valve calcification by two full decades.

Appearance of Calcifications. Four patterns of calcification could be distinguished. These were termed: (1) punctate; (2) blocky; (3) double; and (4) incomplete

Table II

INCIDENCE OF CALCIFICATIONS AT VARIOUS SITES IN THE HEART IN RELATION TO AGE

Age (yr.)	No Calcification	No. of Hearts Showing Calcification*			
		Coronary	Aortic Valve	Mitral Valve	Other
40 or less	3	2	1	0	0
41–50	1	4	1	1	0
51–60	2	10	1	0	0
61–70	5	13	4	3	0
71–80	1	18	8	4	1†
81–90	1	12	7	2	2†‡
91–100	0	2	2	1	0
	13	61	24	11	2

* Hearts showing calcification at two sites are entered twice in this table.
† Calcified annulus fibrosus.
‡ Epicardial calcification secondary to tuberculous pericarditis.

double. Figure 1 demonstrates an example of each type of calcification.

(1) Punctate lesions were discrete and occurred in various shapes: round, oblong, crescentic, and irregular. They varied in size from less than 1 mm. to over 5 mm. They were usually too small for a single lesion to indicate the course or size of a vessel, but if multiple punctate lesions occurred in the same vessel, they suggested its course. When lesions of varying size occurred in the same vessel, the more distal were smaller. When air filled the lumen of an artery adjacent to a punctate calcification, it demonstrated that these lesions extended only part way around the vessel. It therefore appeared that punctate lesions were calcific plates in small atheromas seen in silhouette at various angles.

(2) Blocky calcifications were oblong or linear densities sufficiently large to outline the vessel in which they occurred, indicating its size and direction. They appeared to fill the vessel completely and were as dense in the center as at the edges. They varied from 10 mm. to 30 mm. in length and approximately half were accompanied by other forms of calcification. Their appearance suggested that they were a larger form

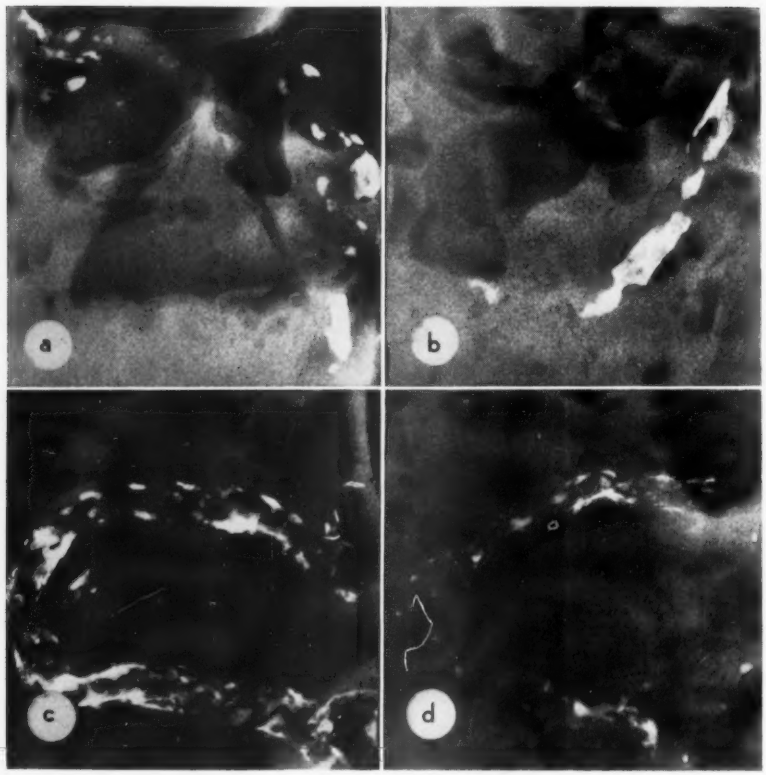

Fig. 1. Four types of calcification found in coronary arteries. (*a*) Punctate lesions at the origin of the left and right coronary arteries. The lowest calcification of the group at upper left was in the aortic valve. (*b*) A blocky calcification of the left anterior descending coronary artery. (*c*) Double calcification of the right coronary artery. (*d*) Incomplete double calcification of the right coronary artery.

of punctate lesions which completely encircled the arterial wall.

(3) Double calcifications represented a series of moderately large, closely-spaced punctate lesions outlining both sides of the vessel, but not filling it completely. The lesions were not confluent, but were often joined by irregular fine connections. The course of the vessel and its external diameter could be judged from these lesions. One double calcification was observed to be 120 mm. in length. A heart with all three coronaries outlined by double calcification is shown in Figure 2.

(4) Incomplete double calcifications were the least common type encountered and the most difficult to see. The vessel was faintly outlined on one or both sides by small, widely-separated, unconnected lesions, usually about 1 mm. in diameter. These suggested the course of the vessel and indidicated its external diameter.

Size of Calcifications. The predominant lesion in each artery exhibiting calcification was classified. When punctate lesions predominated (67 arteries), the largest lesion was measured and the others counted. In 19 of the 67 arteries (28 per cent), the largest lesion was 1 mm. or less in diameter. Thirty arteries (43 per cent) contained lesions either 2 or 3 mm. in diameter and the remainder showed larger lesions. The visibility of calcification in arteries having punctate lesions was dependent not only on the size of the lesions, but also on the number present. As a rule, two to four occurred in each artery. Table III gives data on the size and number of punctate lesions.

When blocky or double calcifications predominated, their length was measured. As indicated by Table IV, the length attained by many of these lesions makes it surprising that they are not seen more frequently on fluoroscopy.

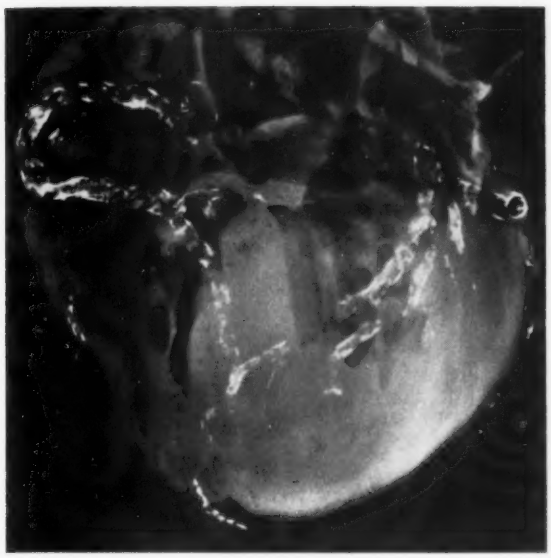

Fig. 2. A heart with all three coronary arteries outlined by double calcification.

Table III

THE SIZE AND AVERAGE NUMBER OF PUNCTATE LESIONS IN A SINGLE CORONARY ARTERY

Size of Largest Punctate Lesion in Artery	No. of Arteries Having Largest Punctate Lesion of Given Size	Average No. of Punctate Lesions in Artery Having Largest Lesion of Given Size
1 mm. or less	19	3.3
2 mm.	15	2.6
3 mm.	15	2.5
4 mm.	6	4.0
5 mm.	8	3.1
6 mm.	2	
7 mm.	1	
8 mm.	0	
9 mm.	0	
10 mm.	1	

Incomplete double calcifications were difficult to see. Their visibility appeared to depend on a complex relationship between total length of the calcification, individual lesion size, and the pattern formed by individual lesions. Measurements are not given for calcifications of this type. Their length was of the same general order as that of double calcifications.

To investigate the error involved in estimating lesion size from roentgenograms made of the intact heart, the following experiment was performed. From 20 hearts, the intact coronary arteries were dissected free, laid flat on film, and re-roentgenographed. Without exception, measurement of punctate lesion length made on roentgenograms of the intact heart was shown to be correct. However, a variable foreshortening was found to occur in blocky, double, and incomplete double calcification, because their long axis did not always parallel the roentgenographic film.

Location of Calcifications. Calcifications were most frequently encountered in the proximal part of the coronary arteries, usually within 2 cm. of the root of the aorta (this was identified by the air shadow it contained). Calcifications of the peripheral branches of the coronary artery, without involvement of the proximal parts, were not seen. Calcific areas sometimes extended to the periphery of a vessel (particularly double calcifications) but only when central areas were also involved.

A line drawn half-way between the apex of the heart shadow and the root of the aorta was never found to have calcification within a coronary artery lying below it un-

Table IV

LENGTH OF BLOCKY AND DOUBLE CALCIFICATIONS

Length of Lesion	No. of Arteries Showing Lesions of Given Length	
	Blocky	Double
(mm.)		
6–10	4	2
11–15	3	1
16–20	1	2
21–25	1	4
26–30	3	3
30–35		2
36–40		1
41–45		4
46–50		2
50–55		3
65		1
90		1
120		1

TABLE V

DISTRIBUTION OF THE VARIOUS FORMS OF ARTERIAL CALCIFICATIONS

Artery	No. of Arteries Showing Calcification				
	Punctate	Blocky Double		Incomplete Double	Totals
Left	35	10	9	3	57
Circumflex	22	3	10	2	37
Right	10	1	15	2	28
Total	67	14	34	7	122

less calcification was also present above it. Calcified coronary arteries occurred exclusively above this line in 58 per cent of hearts and crossed this line in 42 per cent of hearts.

Distribution of Calcifications. The distribution of various forms of calcifications is shown in Table v. The left coronary artery was the most commonly involved, the right coronary artery the least commonly involved. Punctate lesions were most frequent in the left coronary and circumflex arteries. Double calcification was most frequent in the right coronary artery.

DISCUSSION

It seems from this and previous studies that the incidence of coronary calcifications found at autopsy greatly exceeds that demonstrable during life. Pyke and Symons[3] estimate that only 1 of 8 intracardiac calcifications will be discovered by fluoroscopy *in vivo*. Coronary calcifications found by fluoroscopy and illustrated by previous workers appear to be of the type we term "double" or "blocky" and are approximately 40 mm. or more in length. If this length is taken as the minimum extent of calcification required to be visible on fluoroscopy, 1 of 9 hearts in our series with calcification could have been seen. The most common coronary calcification is either 1, 2, or 3 mm. long, and is one of a group of 2 to 4 which lie in the artery close to the root of the aorta. Such calcifications are not seen during life. However, when the excised heart is roentgenographed, they cast shadows as dense as valvular calcification which can be seen *in vivo*. In addition, the majority of these lesions are as large as other lesions which can be seen elsewhere in the chest. In the lung fields, solitary nodules which exceed 3 mm. in diameter can be recognized.[5] More than half of coronary calcifications occur in a group containing at least one lesion exceeding 3 mm. in size.

The apparent invisibility of the majority of coronary calcifications appears due to at least three factors: (1) interference from shadows cast by the spine and ribs; (2) interference from radiation scattered by other structures in the chest; and (3) blurring from the motion of the beating heart. Snellen and Nauta[6] report that, by proper positioning of the patient, interference from the spine can be avoided. Lenk[2] has shown that diminution of radiation scattering with a Bucky diaphragm greatly improves the visibility of coronary calcifications. To minimize cardiac motion, short roentgenographic exposures have been suggested by several authors.[1,2] However, no extensive effort with simultaneous attention to all three of these factors has been reported.

It is now customary to search for intracardiac calcifications by fluoroscopy. The present study indicates that the majority of coronary calcifications are too small to be seen in motion by the unassisted eye. Either cineroentgenography or extremely short single exposure roentgenograms are required. If the beam is properly directed and scattered radiation minimized, it should be possible to visualize these lesions much more frequently than can now be done.

Present knowledge does not allow prediction as to the clinical value of a technique consistently demonstrating the majority of intracardiac calcifications because only the largest lesions are now seen. There is evidence that calcifications may be associated with stenotic lesions of the coronary

artery.[7] Such lesions are not likely to reach any appreciable size before the patient dies of ischemic heart disease. Similar reasoning suggests the possibility that the large calcifications seen on fluoroscopy may occur in relative benign lesions of the coronary artery which are compatible with prolonged survival. These assumptions are neither confirmed nor denied by a review of the literature describing the pathologic anatomy of coronary calcification. However, such review does indicate that except in infants and in states of generalized calcinosis, discrete visible calcifications occur only in association with atherosclerotic lesions. This suggests that a roentgenogram exhibiting coronary calcification should have value in the clinical diagnosis of coronary atherosclerosis. Since coronary calcification has been shown, by the present study, to appear in patients forty to sixty years of age, there is some promise that a roentgenographic method might be useful at a time of life when it is particularly important to recognize coronary atherosclerosis before it leads to ischemic heart disease.

CONCLUSIONS

1. Sixty-one of 76 adult human hearts roentgenographed post mortem exhibited calcific lesions in the coronary arteries.

2. Coronary calcifications were the most frequently encountered intracardiac radiopaque lesions and were more commonly present than absent in patients over forty years of age.

3. The most common calcific coronary lesions were 1, 2, and 3 mm. punctate densities which occurred in groups of 2 to 4 in the proximal portions of the artery.

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CINECARDIOMETRY AND CINE-ELECTROKYMOGRAPHY*

NEW ANGIOGRAPHIC TECHNIQUES FOR THE ANALYSIS OF CARDIAC MECHANICS

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THE purpose of this paper is to describe a cineroentgenologic method of representing graphically details of cardiac mechanics and hemodynamics to be called *cinecardiometry*, and to introduce a discussion of its clinical and experimental application.

In an effort to follow modern cardiovascular investigations stimulated by the progress of cardiac surgery, several years ago[4,5] we started an analytical study of the cineangiocardiograms.

High speed cineangiocardiography[14,19] had introduced a new diagnostic element for recording cardiac dynamics. Only after the development of the analytical projector[18,20] were we able to refine our usual gross or *qualitative* appraisal of these motion picture sequences and to conceive a method for graphic *quantitative* analysis of the structural and hemodynamic events demonstrated in cineangiocardiograms.

The need for understanding cardiac mechanics and hemodynamics has been felt since the beginning of modern cardiac physiopathology. The advent of cardiac catheterization provided a satisfactory means for investigating hemodynamics. However, cardiac mechanics is still a problem, although many methods have been devised to study it. Fluoroscopic observation of the cardiac motion is obviously limited.[15] Roentgenkymography and electrokymography have failed to achieve wide application because of the difficulties created by overlapping anatomic structures.[15] Fast angiocardiography provides a much more satisfactory means of evaluating shape, size and function of the heart chambers and of the individual structures at various phases during the cardiac cycle. Furthermore, the contrast method adds the possibility of studying the dynamics of blood flow by permitting the measurement of contrast density changes in vessels and chambers during the cardiac cycle.

Area and density changes in chambers or vessels have already been reported from experimental angiocardiograms in humans[12] and cineangiocardiograms in dogs.[16] The importance of these studies is relative because cardiac cycles have been recorded only by a few seriograms or frames. The value of any roentgenologic method directed at an analysis of the motion of chambers and great vessels is automatically enhanced by increasing the rapidity or frequency in serial filming during the heart cycle. This advantage is epitomized currently in cineangiocardiography, which can reproduce scenes lasting through several cardiac cycles at up to 30 frames per second in adults and 60 frames per second in children.

MATERIAL AND METHOD

The technical details of cineangiocardiography have already been described elsewhere.[14,19]

Cinecardiometry is a cineroentgenologic method of representing graphically the motion of the cardiovascular anatomic structures opacified by contrast medium. This term is proposed to distinguish this method from "roentgenkymography" and "electrokymography."

Utilizing the analyst projector, each

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frame of the cineangiocardiogram is projected in sequence and the borders of the great vessels and cardiac chambers are traced precisely. Each important anatomic structure visible in every frame is marked as a reference point, and the events of the cardiac cycle, such as the opening of the semilunar valves and rapid filling of the ventricles, are noted on the numbered drawings. Timing is accurately recorded by marking the sequential number of each frame. This is possible because the cinefluorocamera is driven by a motor synchronized with a 60 cycle current and is also connected with an electrocardiograph which records the exposure time of each frame.* Next, using the various anatomic reference points, representative diameters are traced and measured. The areas of the projected silhouettes of the cardiac chambers are determined by a planimeter.

The measurements of a given diameter in millimeters or a given area in square centimeters are listed chronologically. However, these measurements cannot be transposed directly onto graph paper because they contain errors due to geometric distortion and magnification produced by the short distance (26–36 inches) at which the cineangiocardiograms are recorded. Such errors are difficult to calculate. Nevertheless, they may be reduced to acceptable limits and ignored completely when each measurement is expressed simply as a percentage of the arithmetic average of the whole group of measurements recorded for each diameter or area.

The percentages, transposed chronologically to the graph paper, permit the drawing of continuous curves against time as abscissa. The baseline of each curve is the arithmetic average of measurements from several cardiac cycles and arbitrarily is made equal to 100 per cent. This average is theoretically the size of the heart structures at the mid-point between systole and diastole when they are in the resting phase. This means that each curve expresses in percentage form the dynamic variation of the particular stucture from its static size. These curves are comparable to each other not only in the same subject, but also in different subjects because they represent only the percentage variation of the dimension of the cardiac structure and are independent of true size.

This method has been applied to the study of the circumference and cross section of the great vessels and to the areas and volumes of the atria and left ventricle. In calculating approximate volumes, it is assumed that the two transverse diameters of these chambers are equal and that their shape is an ellipsoid. The formula, Volume $= 4/3\pi abc$ in which a, b and c are the semi-axes of an ellipsoid has been adapted as follows: Volume $= 4/3\pi ab^2$ in which a is the longitudinal semiaxis and b the transverse semiaxis of the silhouette of these heart chambers. The volume of the right ventricle cannot be calculated from a cineangiocardiogram made in one projection because of its complicated three dimensional representation in any of the usual angiocardiographic projections.

Cine-electrokymography is an electrokymographic method of recording from the cinefluorogram projected on the screen the motion and the density changes of the cardiovascular structures opacified or not by contrast material.

A Sanborn electrokymograph has been adapted to react directly to the light of the film projector. The over-all brilliance of the projected image is adjusted by an iris diaphragm placed on the objective and by photographic filters placed on the slit of the photocell. A carbon-brush contact on the projector gear, connected to the "remote marker" of the electrocardiograph, is used to record the projection of each frame on

* *Electrocardiographic Correlation.* Electrocardiographic tracings are recorded during the cinefluorographic procedure.

A carbon-brush contact is placed on a gear of the cinecamera and connected to the panel control of the cinefluorographic unit and to the "remote marker" of the electrocardiograph. Only when the roentgenographic tube is energized and the shutter of the cinecamera reaches full opening does the carbon brush contact close the circuit of the "remote marker" of the electrocardiograph. In this manner the exposure time of each frame is recorded by small marks at the bottom of the electrocardiographic tracings. The correlation of each frame to the electrocardiographic tracing permits the correlation of the latter to the cinecardiometric, cine-electrokymographic and densitometric curves.

the lower margin of the electrocardiographic paper band. These marks permit the timing of the cine-electrokymographic tracings.

The film is projected at the rate of 8 or more frames per second; slower speeds may produce a projection flicker which detracts from the value of the tracing. The paper band of the electrocardiograph is run at the rate of 10 or 25 mm. per second. A projector-screen distance of about 3 feet is maintained in order to reproduce the images at the same size in which they appeared on the fluorescent screen. The records obtained from the same anatomic structure may vary slightly in length because the analytical projector is run by an asynchronous motor. The correlation, however, of the tracings recorded from the same film is always possible by means of the above described marks at the bottom of the tracings.

The film used for cine-electrokymographic study must be technically perfect in exposure, processing and printing and the projector must be supplied by a rectified, stabilized current.

Densitometry is an electrophotometric method of recording the optical density of the images of the cardiovascular structures opacified by contrast material.

The films are projected frame by frame. The photocell of the photometer is placed within a small window on the projection screen, and by moving the projector, the image of the anatomic structure is aimed at the photocell. The optical density of the cardiovascular structure prior to the passage of the contrast material is used as the "reference zero." Density changes due to cardiac and respiratory phases must be considered to obtain the "reference zero" or baseline.

Each density measurement is plotted against time on the graph paper, and by joining these points, continuous curves can be drawn.

The curves representing density changes of each cardiovascular structure correlated with time show the progression and distribution of the contrast material within heart chambers and great vessels during each phase of the mechanical cardiac cycle.

APPLICATION: SELECTED EXAMPLES

The following examples are presented to illustrate the application of our methods to the study of cardiovascular physiopathology. The results of extensive studies will be reported in the near future (*see addendum*).

The effects produced by intravenous injection of contrast medium on the normal right heart are illustrated in the cine-cardiometric tracings shown in Figures 1 and 2. The flow through the superior vena cava starts to increase 1.5 seconds after the beginning of injection of medium into the left arm vein. The caval diameters (Fig. 1 and 2; tracings 1, 2 and 3) at the third second have augmented approximately from 80 per cent to 120 per cent. The atrial diameters (Fig. 1 and 2; tracings 4 and 5) have also increased, but apparently not in proportion to the caval diameters. A proportionality does become evident when the caval tracings are compared to that of the atrial volume, calculated from the two main atrial diameters (Fig. 2; tracings 1, 2, 3 and B).

We realize that the superior vena cava is a collapsible vessel, subject to the pressure changes of the mediastinum and that its cross section probably is not circular. However, the relationship of its cross section with the atrial volume was constantly demonstrated in the cases studied.

It has been found in dogs that the injection of contrast material into the right atrium or into the inferior vena cava increases the atrial pressure very slightly (1 or 2 mm. Hg)[13] and that no pressure changes occur in the right heart chambers when the medium is injected into the brachial veins.[11] We observed the same pressure changes in our experimental work in dogs.

It has been emphasized that small pressure changes in the right atrium may be associated with large variation of the cardiac output.[8,10] We consistently ob-

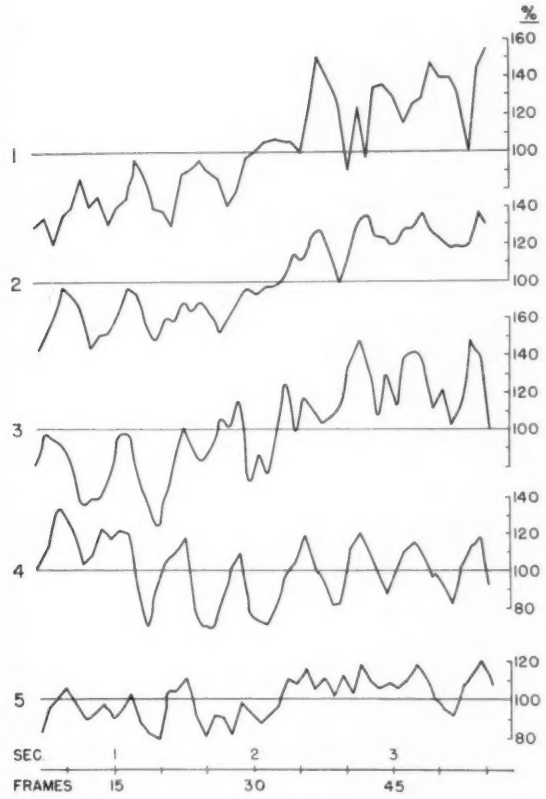

Fig. 1. Cinecardiometric tracings from left anterior oblique cineangiocardiogram recorded at 15 frames per second in a normal five year old subject, showing the effect of peripheral injection of contrast medium on the diameters of the superior vena cava (1, extrapericardial portion, immediately below the azygos vein; 2, intrapericardial portion; 3, caval-atrial junction) and on the main diameters of the right atrial silhouette (4, transverse; 5, longitudinal). Timing is referred to the beginning of the injection. The ordinate scales indicate the percentage variation of each diameter from its average value which has been set equal to 100 per cent. The injection ended after 2.5 seconds. Enlargement of caval and atrial diameters appears 1.5 seconds after the beginning of the injection.

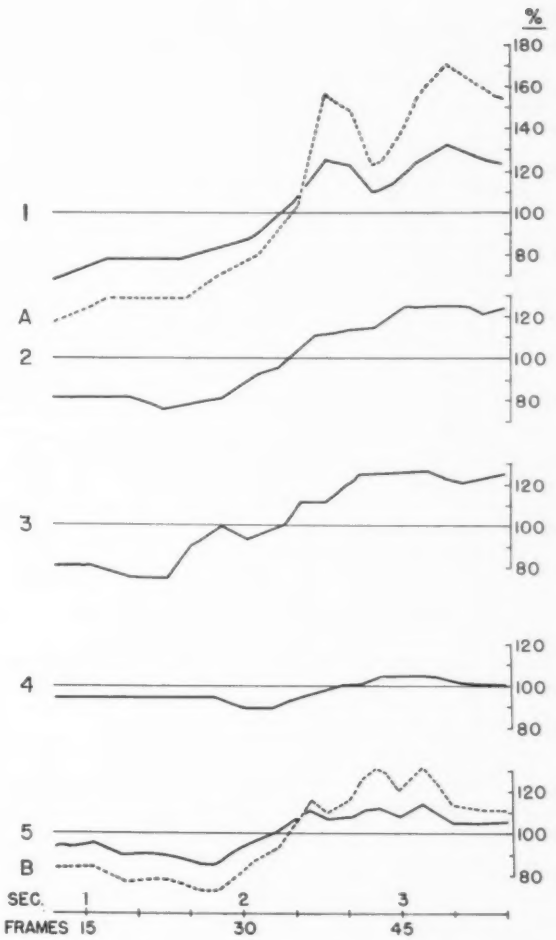

Fig. 2. Composite of curves in Figure 1. The midpoints of the ascending and descending branches were connected. The dotted line tracings have been obtained by the same procedure from curves expressing the variation of the cross sectional area of the extrapericardial portion of the superior vena cava (A) and of the atrial volume (B), respectively, calculated from their diameters. This represents the changes in size of the superior vena cava and of the right atrium independently from systolic-diastolic movements. The changes of the atrial volume are of the same magnitude as those of the caval diameters.

served in the cineangiocardiograms recorded in humans that the silhouette size of the superior vena cava, the right atrium and ventricle and the pulmonary artery increased progressively at each cardiac cycle in proportion to the flow of contrast medium. In addition to the physiologic implication, these facts must be considered during the evaluation of (a) the size of the right heart chambers and (b) the amount and direction to the shunt through the atrial or ventricular septal defect demonstrated by cineangiocardiography.[7]

Alterations in both the mechanics and the hemodynamics of the heart are shown in Figure 3 which demonstrates by cinecardiometry the changes of the left heart occurring during the left angiocardiogram. The patient was ten years old with clinical and cineangiocardiographic diagnosis of aortic

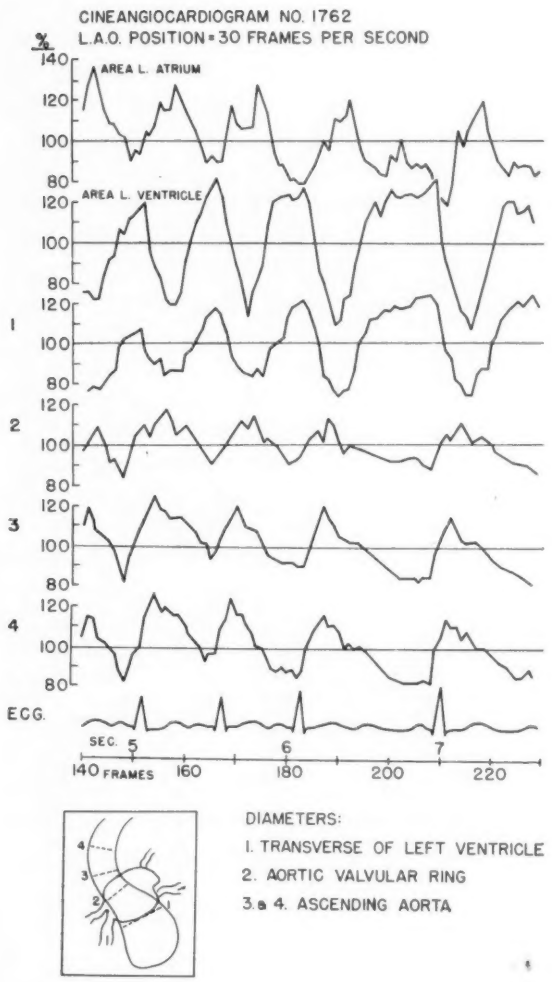

FIG. 3. Cinecardiometric tracings demonstrating the effect of peripheral angiographic injection on the left heart, from left anterior oblique cineangiocardiogram recorded at 30 frames per second in a ten year old patient with minimal stenosis and insufficiency of the aortic valves. Lead II ECG recorded synchronously with the cineangiocardiogram shows 6.5 seconds after the beginning of the injection the absence of QRS complex. This was probably due to a transient atrioventricular block produced by the coronary circulation of contrast medium. The cinecardiometric tracing of the area of the left atrial silhouette shows an abnormally small fifth atrial complex (frame 200) occurring in the absence of the corresponding ventricular systole. The area and the transverse diameter (1) of the left ventricle, during the long lasting diastole due to the atrioventricular block, progressively increase while the aortic diameters (2, 3, and 4) decrease. The sixth atrial complex, preceded by ventricular systole, appears normal. This demonstrated that the atrial filling is principally due to the *vis a fronte* generated by the ventricular ejection (descent of the atrioventricular junction). The left heart silhouette, at the left bottom, shows the diameters studied. Timing in seconds is referred to the beginning of the injection. The ordinate scales indicate the percentage variation of each diameter or area from their average value which has been set equal to 100 per cent.

valvular stenosis. These tracings represent dimensional changes of the left heart chambers and of the aorta in detail, since each cardiac cycle was recorded on 17 frames. The lead II electrocardiogram, recorded synchronously with the cineangiocardiogram at a speed of 50 mm. per second, shows the absence of QRS complexes 6.5 and 7.25 seconds after the beginning of the injection of contrast material into the arm vein. Since the only tracing obtained was in lead II, the most probable diagnosis seemed to be a transient atrioventricular block probably due to myocardial anoxia produced by coronary circulation of contrast medium. However, the cinecardiometric tracings show (Fig. 3) that the fourth atrial complex is not followed by ventricular contraction. The fifth cardiac cycle presents an abnormally small atrial complex. During the long lasting ventricular diastole, due to the transient atrioventricular block, the area and the transverse diameter of the left ventricle increased. This demonstrated that the atrial filling is mostly due to the aspirating force (*vis a fronte*) generated by the descent of the atrioventricular junction during ventricular systole.[1] The sixth atrial complex which is preceded by a regular ventricular contraction is, in fact, normal. Comparison of dimensional changes of the transverse diameter and of the area of the left ventricle, which occur during the fourth ventricular complex, demonstrates that the transverse diameter of the ventricle increases during the long lasting diastole.

Cine-electrokymographic tracings, recorded on the same cineangiocardiogram, show the role played by the different parts of the left ventricle during the cardiac cycle.

The tracings shown in Figure 4 have been

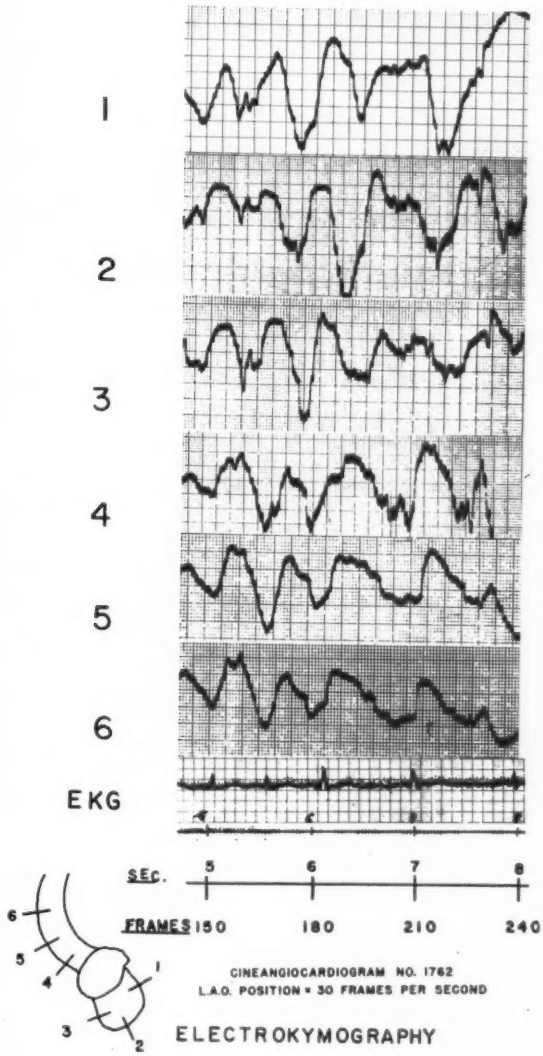

FIG. 4. Cine-electrokymographic tracings recorded on the left ventricular walls (1, 2 and 3) and on the ascending aorta (4, 5 and 6) from the same cineangiocardiogram as Figure 3. Correlation against time and lead II ECG, recorded synchronously with the cineangiocardiogram, was made by the photographic method because of the different lengths of the records (see "Material and Method"). The ECG tracing has been retouched by ink to render it more visible on the photographic reproduction. Timing is the same as in Figure 3. The silhouette at the bottom of the figure shows the positions of the recording photocell slit of the electrokymograph. The records demonstrate the similarity of cinecardiometry and cine-electrokymography. The motion of the external walls (1), tip (2) and septum (3) of the left ventricle are shown.

correlated with each other and with time by a photographic method. We could not obtain the same length in all the tracings originally recorded because the analytical projector was not run by a synchronous motor. However, the curves of the fourth cardiac cycle (Fig. 4; tracings 1, 2 and 3) show that the left ventricle increases in length during the phase of "rapid ventricular filling." In presystole, the ventricular length decreases slightly while the transverse diameter (tracings 2 and 3) increases in proportion.

By comparing diameter 1 (Fig. 3) and tracing 1 (Fig. 4) the similarities of the cinecardiometric and the cine-electrokymographic tracings become evident. The aortic tracings of both Figures 4 and 5 also compare favorably.

Tracings of angiographic density prove to be very helpful in studying hemodynamics, especially when correlated with cinecardiometric or cine-electrokymographic tracings. For instance, the angiographic density of the pulmonary artery depends upon the flow (concentration and rate of injection) of contrast material injected. The systolic-diastolic variation of this density is very slight.

This is demonstrated in Figure 5 in which the pulsation of the pulmonary artery (cine-electrokymogram; tracing at the top) and density changes (cine-densigram; tracing at the bottom) have been recorded in the same area from a case of pulmonary valvular stenosis. When this disease is complicated by patent ductus arteriosus, the density of the pulmonary artery greatly diminishes during diastole. Figure 6 shows the density changes of pulmonary artery (tracing at the top) compared with the pulsation of the left ventricle (cine-electrokymogram; tracing at the bottom) to demonstrate that the loss in density occurs in diastole. This cyclic, diastolic loss of angiographic density of the pulmonary artery is due to the dilution of contrast medium produced by the shunt of nonopaque blood coming from the aorta via the ductus. This

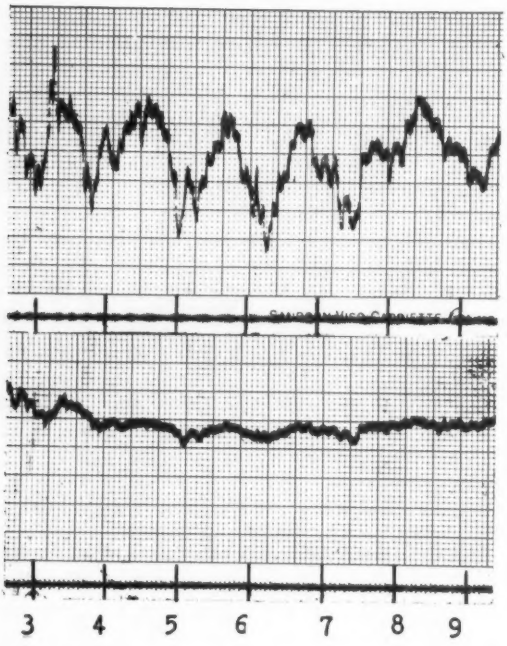

FIG. 5. Pulsation (cine-electrokymogram at the top) and angiographic density changes (cine-densigram at the bottom) recorded in the same area of the pulmonary artery from a 30 frame per second left anterior oblique cineangiocardiogram. The patient had uncomplicated pulmonary valvular stenosis. Timing in seconds at the bottom of each tracing is referred to the beginning of peripheral angiographic injection. The injection was unusually long (6 seconds). This figure demonstrates that the angiographic density of the pulmonary artery varies slightly during the cardiac cycle (upward deviation = increase in density.)

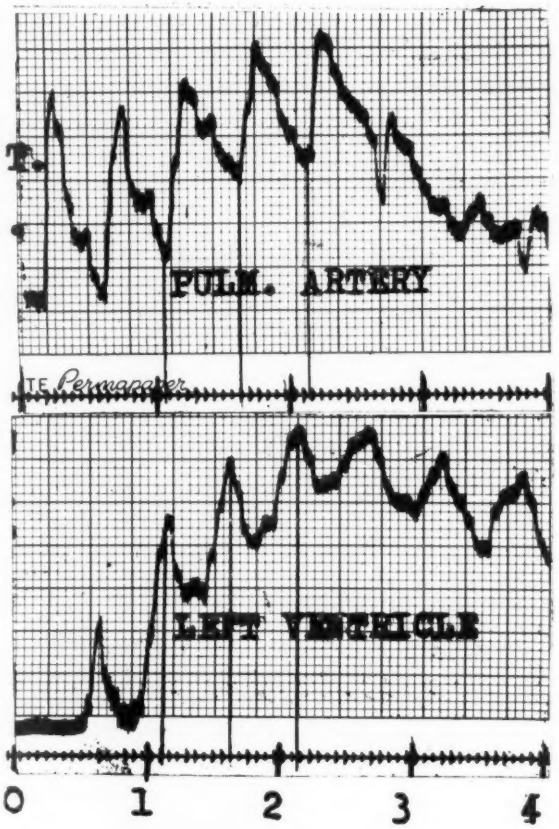

FIG. 6. Angiographic density of the pulmonary artery (cine-densigram at the top) and pulsation of the left border of the left ventricular silhouette (cine-electrokymogram at the bottom) recorded from a 15 frames per second left anterior oblique cineangiocardiogram. The patient was five years old and had pure pulmonary valvular stenosis complicated with patent ductus arteriosus. The timing of each tracing is in seconds. The peripheral injection of contract medium ended at the first second. The baseline of the ventricular cine-electrokymogram is ascending because of the increased angiographic density of the lungs produced by the contrast medium circulating through the pulmonary vascular bed. This figure demonstrates that during the right angiocardiogram the angiographic density of the pulmonary artery decreases greatly in diastole (blanching sign of the patent ductus arteriosus). This is due to the dilution of contrast medium produced by the nonopaque blood coming from the aorta via ductus. (The tracings have been retouched by ink to render them more visible on the photographic reproduction.)

cineangiographic sign was first reported by members of this department[9] and has been extensively described by the author.[3,6]

Figure 7 shows that the lower part of the outflow tract of the right ventricle (tracing 4) does not contract simultaneously with the ventricle (tracing 5). During the first part of systole, this portion is wide open and contracts only at the end of ventricular ejection. The middle portion of the infundibulum (tracing 3) bulges during the first part of systole and contracts later than the lower portion. The subvalvular part (tracing 2) closely follows the dynamics of the pulmonary artery (tracing 1) in which dilatation and contraction are much more delayed.

Our extensive studies have demonstrated that the infundibular contraction is retarded in proportion to the increment of the stroke volume. We found that when the peripheral injection of contrast medium

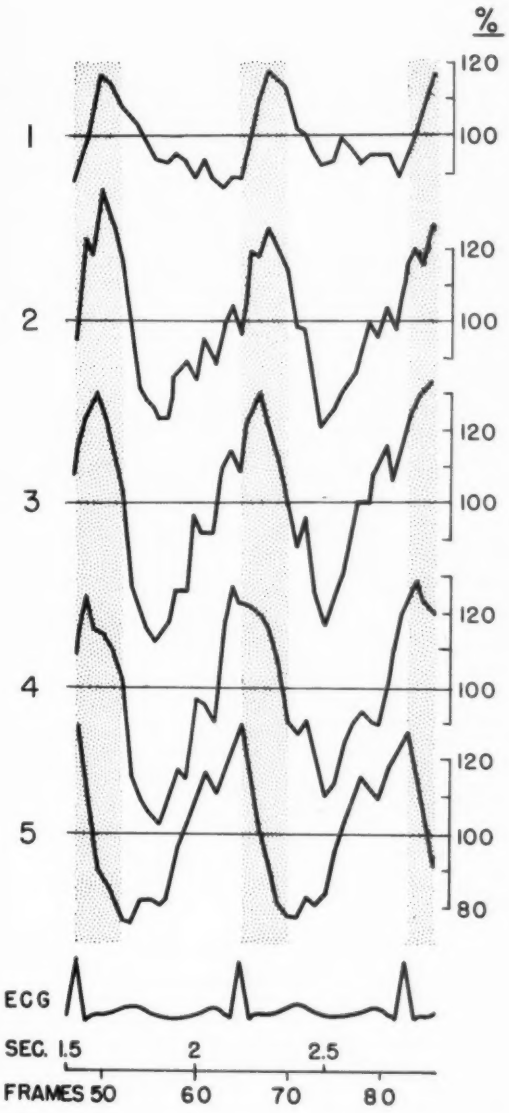

Fig. 7. Cinecardiometric representation of the mechanics of the outflow tract of the right ventricle, from a left anterior oblique cineangiocardiogram recorded at 30 frames per second in a ten year old patient presenting a normal right heart. Diameter changes of the pulmonary artery (1), of the subvalvular (2), mid (3), and lower portions (4) of the outflow tract have been correlated with area variations of the silhouette of the right ventricle (5). The tracings are also correlated with lead II ECG synchronously recorded during the cineangiocardiogram. Ventricular ejection is vertically marked by the zip-a-tone paper. This demonstrated that during the first part of ventricular systole the outflow tract bulges, mostly in its mid and subvalvular portions. Contraction of the outflow tract starts in the second part of systole but the end of contraction is progressively delayed in the distal portions. The contraction of the outflow tract lasts until the next opening of the atrioventricular valves (end of T wave). Note the similarity of the complexes of the pulmonary artery and of the subvalvular portion. Timing in seconds is referred to the beginning of peripheral angiographic injection. The scales on the ordinate indicate the percentage variation of each diameter or area from their average values which have been set equal to 100 per cent.

augmented the venous return the areas of the right atrial and ventricular silhouettes and the diameters of the infundibulum and pulmonary artery were increased, while the contraction of the outflow tract was proportionally delayed.

This finding has been applied to the cineangiocardiographic differential diagnosis of congenital and acquired diseases involving the outflow tract of the right ventricle and the pulmonary valves.[2,17]

CONCLUSION

The tracings recorded by our method are reproducible and comparable. They represent graphically the mechanics and the hemodynamics of each cardiovascular structure in every phase of the cardiac cycle as recorded by cineangiocardiography.

The characteristics of each curve may vary in different subjects in some details depending upon: the number of the frames recording each cardiac cycle; the position in which the cineangiocardiogram was recorded; the photographic qualities of the film obtained; the injection of contrast medium (concentration and rate of injection).

Cinecardiometric curves are similar to the electrokymographic tracings because they also represent the motion of the cardiovascular structures studied. While electrokymography records border motion of the heart chambers and great vessels as observed by fluoroscopy, the cinecardiometric method records more precisely the variations of diameters, cross sections, areas and volumes of the same structures, well outlined by contrast material.

The cine-electrokymographic method of

cardiac motion analysis has the advantage of identification of each anatomic structure and a complete motion analysis of different structures from the same movie record. This method may be enhanced by introduction of a gas contrast medium into the mediastinum or into the pericardial cavity.

Cinecardiometric records of the cardiac mechanics and the angiographic densitometry of hemodynamics can represent the specific function, normal or pathologic, of every anatomic structure during the cardiac cycle.

This representation is enhanced when these records are correlated with each other and with electrocardiograms, pressure tracings, pneumograms, etc., recorded simultaneously with the cineangiocardiogram.

Under these conditions, cineangiocardiographic interpretation is no longer a subjective study, but the result of an objective analysis of the iconographic and dynamic elements of the examination or, in other words, of the shape and function.

The method, obviously, is not recommended in routine cineangiographic study, but in selected cases or in special investigation of cardiac physiopathology.

SUMMARY

Methods for analyzing cardiac mechanics and hemodynamics directly from high speed cineangiocardiographic records are described.

Cinecardiometry is a roentgenologic method of representing graphically the motion of the cardiovascular anatomic structures opacified by contrast medium. This term is proposed to distinguish it from "roentgenkymography" and "electrokymography." The method consists in drawing precisely the borders of the silhouettes of heart chambers and great vessels directly from the projected images of each frame of the cineangiocardiogram. Diameters and areas are measured chronologically from the drawings. Volumes of the cardiac cavities are calculated. Each measurement of each class of diameters, area and volume, expressed as a percentage of the arithmetical average of the whole class, is plotted on the ordinate against the time. By joining these points, continuous curves are drawn. The shape of these curves is similar to those of the electrokymographic records.

Densitometry is a photometric method for recording the angiographic density of the cardiovascular structures from each consecutive frame of the cineangiocardiogram. By plotting chronologically each value on the ordinate against the time, continuous curves can be traced. Curves recorded from the same film, in different parts of the cardiovascular silhouette and correlated with each other, can represent the progression and the distribution of the contrast medium peripherally injected into the great vessels and cardiac cavities.

Cine-electrokymography and *cine-densitometry* are electrokymographic techniques for recording, from the cinefluorogram projected on the screen, the border motion and the density changes of the cardiovascular structures opacified or not by contrast medium.

Correlation of the tracings recorded by these methods with electrocardiograms, pressures, pneumograms, etc., recorded synchronously with the cineangiocardiogram can represent the cardiac performance in each phase of the cycle.

Illustrative examples are presented and briefly discussed. The applications of these methods to cardiovascular investigation and diagnosis are considered.

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ADDENDUM

Since this paper was accepted the following papers have been published: (1) Campeti, F. L., Gramiak, R., Watson, J. S., and Ramsey, G. H. Functional diagnosis of patent ductus arteriosus studied by cineangiocardiography in fifty-three cases. *Circulation*, 1958, *18*, 887–896. (2) Campeti, F. L., Ramsey, G. H., Gramiak, R., and Watson, J. S., Jr. Dynamics of the orifices of the venae cavae studied by cinenangiocardiography. *Circulation*, 1959, *19*, 55–64.

THE ROENTGENOLOGIC FEATURES OF EBSTEIN'S ANOMALY OF THE TRICUSPID VALVE

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ALTHOUGH Ebstein's anomaly is a relatively rare congenital heart lesion, it is of importance to the radiologist because of its striking roentgenologic features. The correct diagnosis can often be made on the basis of the plain roentgenograms alone. In spite of this only one paper on this subject has appeared in the American radiologic literature.[1]

INCIDENCE

Ebstein's anomaly constitutes a small proportion, probably less than 1 per cent of all congenital heart defects. More than 100 cases have been reported in the world literature. To date we have collected 23 cases of this malformation at the University of Minnesota Hospitals, 8 of which came to autopsy.

PATHOLOGIC FEATURES

Wilhelm Ebstein described in 1866 the characteristic downward displacement of the tricuspid valve from the true annulus. The valve leaflets show a wide pathologic variation. They may be very large, rudimentary or even totally absent. The leaflet material may be adherent to the endocardium. Portions of the valves may be thick and nodular, or may be very thin with multiple perforations.

A half-tone sketch of a typical postmortem specimen is shown in Figure 1. Also shown is a roentgenogram of the same specimen, correlating the anatomic features with the roentgen findings (Fig. 2). To obtain this second view, the left side of the heart was filled with lubricating jelly following which the right atrium and right ventricle were outlined by double contrast technique using barium sulfate and air. This technique helps to demonstrate the severe deformity of the tricuspid valve and the malformed papillary muscles and chordae tendineae. There is also marked thinning of the ventricular musculature and dilatation of the right ventricular outflow tract. The latter accounts for the prominent bulge along the left upper heart contour in the routine chest roentgenogram (Fig. 3).

CLINICAL FEATURES

The clinical features of the 23 patients varied considerably, probably due to the great variability in the degree of deformity of the tricuspid valve. This deformity may result in functional tricuspid stenosis, tricuspid insufficiency or in fairly normal hemodynamics. Dyspnea, excessive fatigue, cyanosis, bouts of tachycardia, and squatting are common complaints and symptoms. Systolic and diastolic murmurs over the precordium, "triple" or "quadruple" rhythms, and a right bundle branch block or Wolff-Parkinson-White pattern on the electrocardiogram are helpful diagnostic features. Typically these patients are mildly cyanotic in the neonatal period, become acyanotic, and then again develop cyanosis in latter childhood or adult life. The cyanosis and decreased pulmonary flow usually result from a right to left shunt at the atrial level through an incompetent foramen ovale. The average life expectancy lies between twenty and thirty years of age.

We have reviewed the clinical, physiologic, and pathologic features of this anomaly in great detail in a separate publication.[2]

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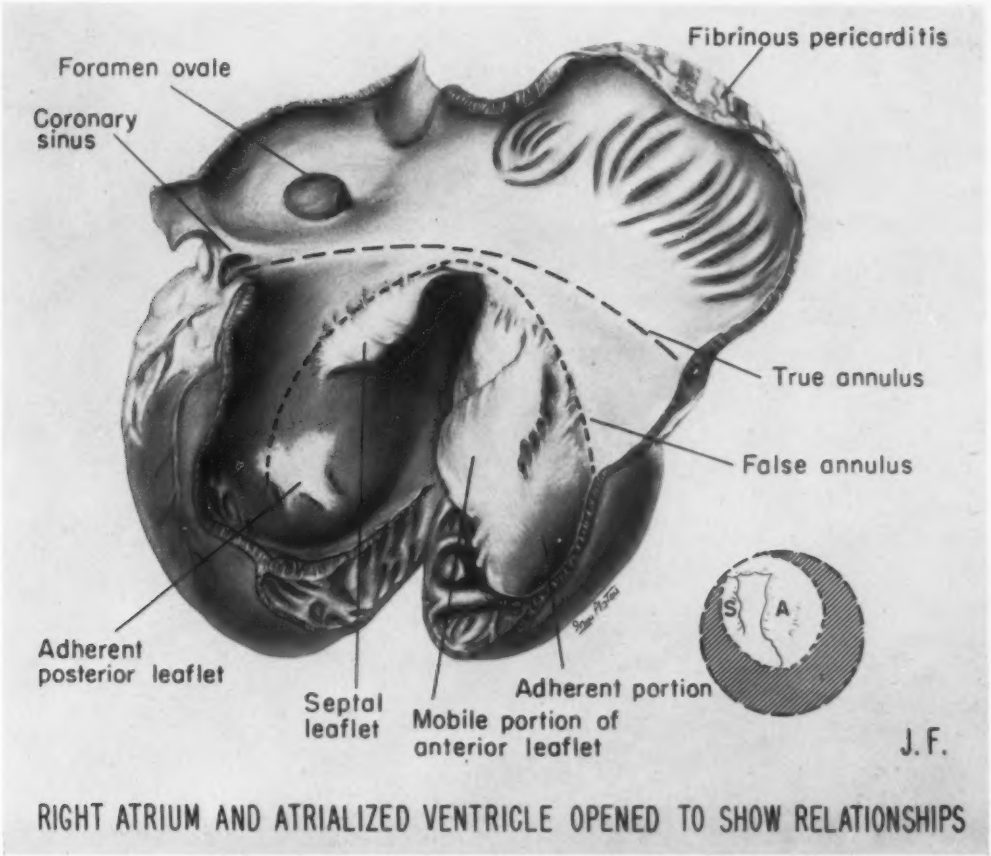

Fig. 1. Half-tone sketch of postmortem specimen.

ROENTGEN FINDINGS

The posteroanterior roentgenograms of our autopsied cases are shown in Figure 4 *A–D* and *E–H*. The most impressive and characteristic feature is marked cardiac enlargement with normal or decreased pulmonary vasculature. The cardiomegaly is due primarily to massive dilatation of the right side of the heart.

In advanced cases the massively dilated right atrium, which may contain several thousand cubic centimeters of blood, may be suspected on the roentgenograms by an increased radius of the "right atrial segment" (Fig. 3). In such cases the right atrium may form the posterior heart contour in the left anterior oblique and in the lateral position (Fig. 5). If there is associated dilatation of the right ventricular outflow tract, the left heart border characteristically bulges almost horizontally towards the left chest wall. This horizontal "take-off" of the left upper heart contour together with right atrial prominence is responsible for the classic "box-like" silhouette of Ebstein's malformation.

In milder cases there may be a "sloping" left heart border suggestive of left ventricular enlargement. In the left anterior oblique and lateral views, the "enlarged left ventricle" is in fact the right atrium which is border-forming in all projections or a normal left ventricle which is pushed backward by the dilated right heart.

It is important to keep in mind that some cases of Ebstein's anomaly may have essentially normal cardiac contours (Fig. 4*F*).

The pulmonary artery is usually hidden behind the dilated right ventricular outflow tract. It is often impossible to visualize a pulmonary artery segment by fluoros-

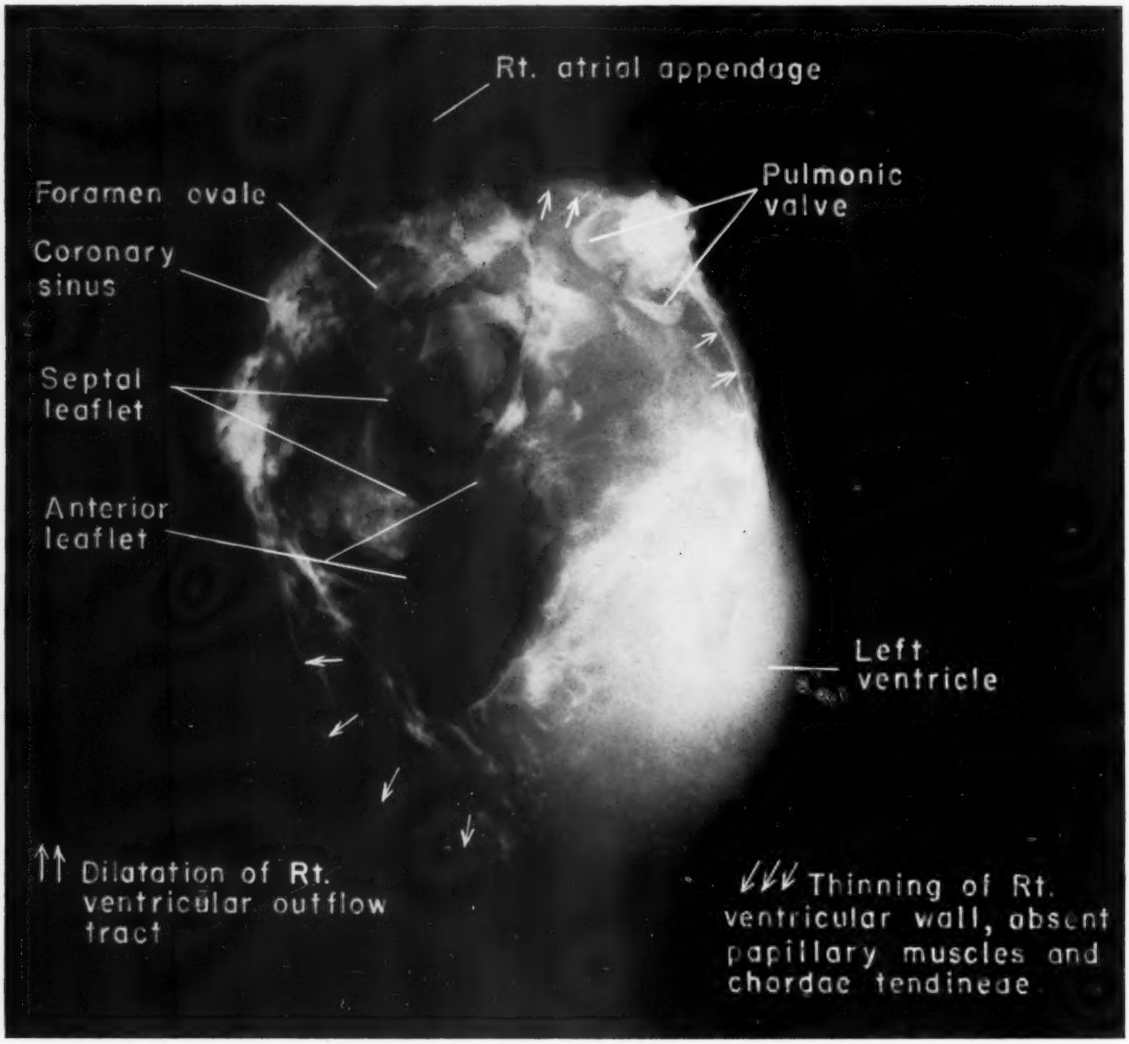

Fig. 2. Roentgenogram of same specimen demonstrates displaced valves, marked thinning of the right ventricular musculature and outflow tract.

copy. The aortic arch may be hidden, but in most cases one is able to demonstrate a normal or relatively small aortic "knob" in the reversed lordotic position.

The marked cardiac enlargement of Ebstein's anomaly is associated with normal or decreased pulmonary vasculature. We have noted an excellent correlation between distinctly decreased pulmonary arterial vasculature and cyanosis. Even in acyanotic patients, the pulmonary vessels tend to be at the lower limits of normal. Pulmonary congestive changes and pleural effusion have been strikingly absent despite massive cardiomegaly and clinical evidence of right heart failure.

Kerley's lines, intracardiac calcifications, and left atrial enlargement have not been observed in any of our 23 cases. The absence of left atrial enlargement is important in the differential diagnosis.

In our experience the pulsations of the right heart border at fluoroscopy or by roentgenkymography were not diagnostic. Pulsations were present in all cases, however, thus helping to exclude pericardial

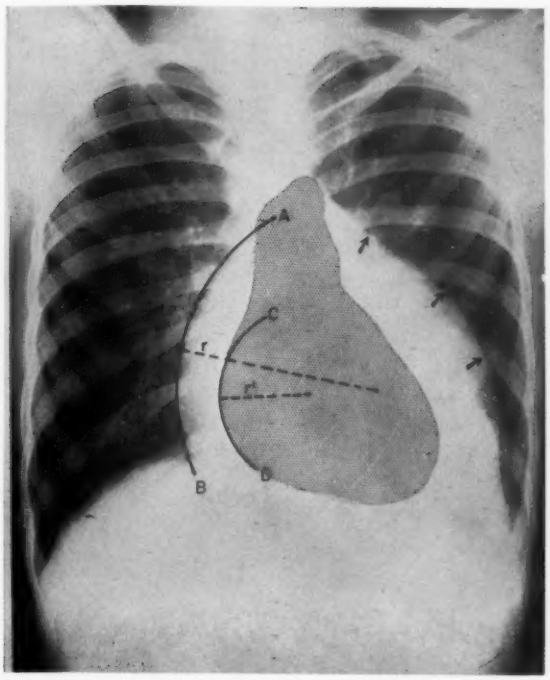

FIG. 3. Roentgenogram of same patient as in Figures 1 and 2. Note dilated right ventricular outflow tract obscuring the pulmonary artery segment making up most of the left border of the heart. Also note increased right "atrial radius" (A–B).

effusion. Often the markedly depressed gastric air bubble showed rather active transmitted pulsations (Fig. 3A).

DIFFERENTIAL DIAGNOSIS

Among cyanotic heart diseases with decreased pulmonary flow, tetralogy of Fallot (pulmonary stenosis with interventricular septal defect), trilogy of Fallot (pulmonary stenosis with interatrial septal defect), and tricuspid atresia may roentgenographically be similar to Ebstein's disease. The massive cardiomegaly of Ebstein's anomaly is rarely encountered in tetralogy of Fallot. The presence in tetralogy of an elevated apex and of an elevated small left ventricle as seen in the left anterior oblique position is a helpful differential diagnostic finding. Trilogy of Fallot and tricuspid atresia usually show evidence of left atrial enlargement.

In some cases a definite differential diagnosis cannot be made by roentgenologic study alone. The clinical information of a harsh systolic murmur and a decreased second sound over the pulmonary area will help to identify congenital heart lesions associated with pulmonary stenosis. Left axis deviation and left ventricular preponderance on the electrocardiogram are a rather consistent finding in tricuspid atresia.

The frontal plane cardiac silhouette in mitral disease may superficially resemble Ebstein's malformation. Nevertheless, left atrial enlargement and accentuated vasculature will, in our experience, definitely exclude Ebstein's anomaly. Massive pericardial effusion can be ruled out by the fluoroscopic findings. In aortic stenosis the most important roentgen feature is a prominent and actively pulsating ascending aorta on fluoroscopy (poststenotic dilatation).

Myopathies and subendocardial fibroelastosis may be differentiated by the electrocardiogram and accentuated pulmonary vasculature (if in failure). Left atrial enlargement characteristically is seen in subendocardial fibroelastosis.

SPECIAL PROCEDURES

We consider cardiac catheterization to be the most satisfactory method of identifying this anomaly, if the clinical diagnosis remains in doubt. The important diagnostic features during catheterization are the localization of the tricuspid valve by pressure changes and a normal or slightly elevated right ventricular pressure. An anteroposterior spot roentgenogram should be taken to document the valve position which is characteristically displaced to the left of the spine. However, one must remember that the tricuspid valve may also be displaced to the left in other conditions associated with marked right atrial dilatation.

The intracavitary electrode catheter can aid in establishing the diagnosis by identifying ventricular musculature proximal to the tricuspid valve in a low pressure area.[3]

In our experience venous angiocardiog-

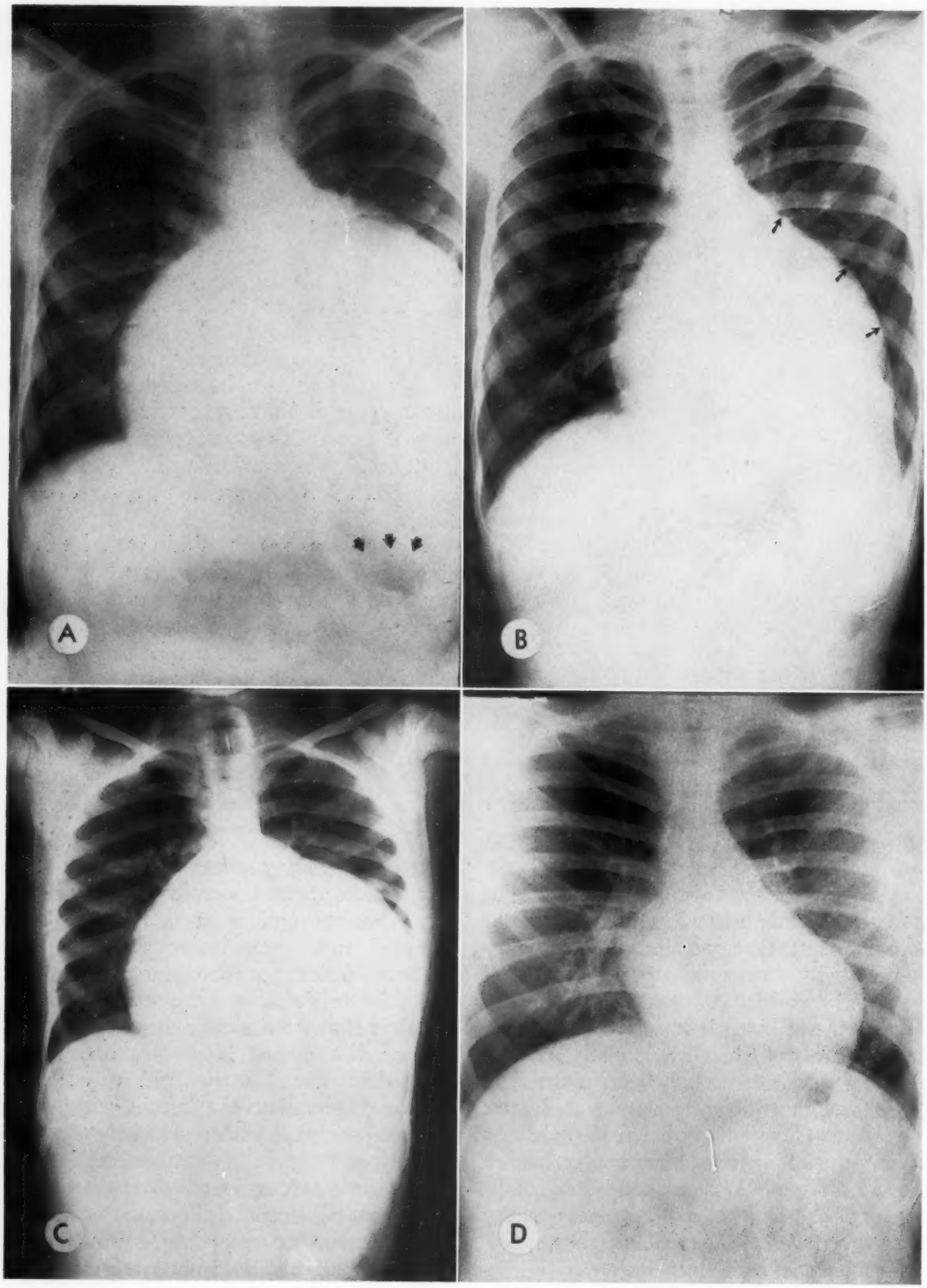

Fig. 4. (*A–D*) Roentgenograms of patients coming to autopsy.

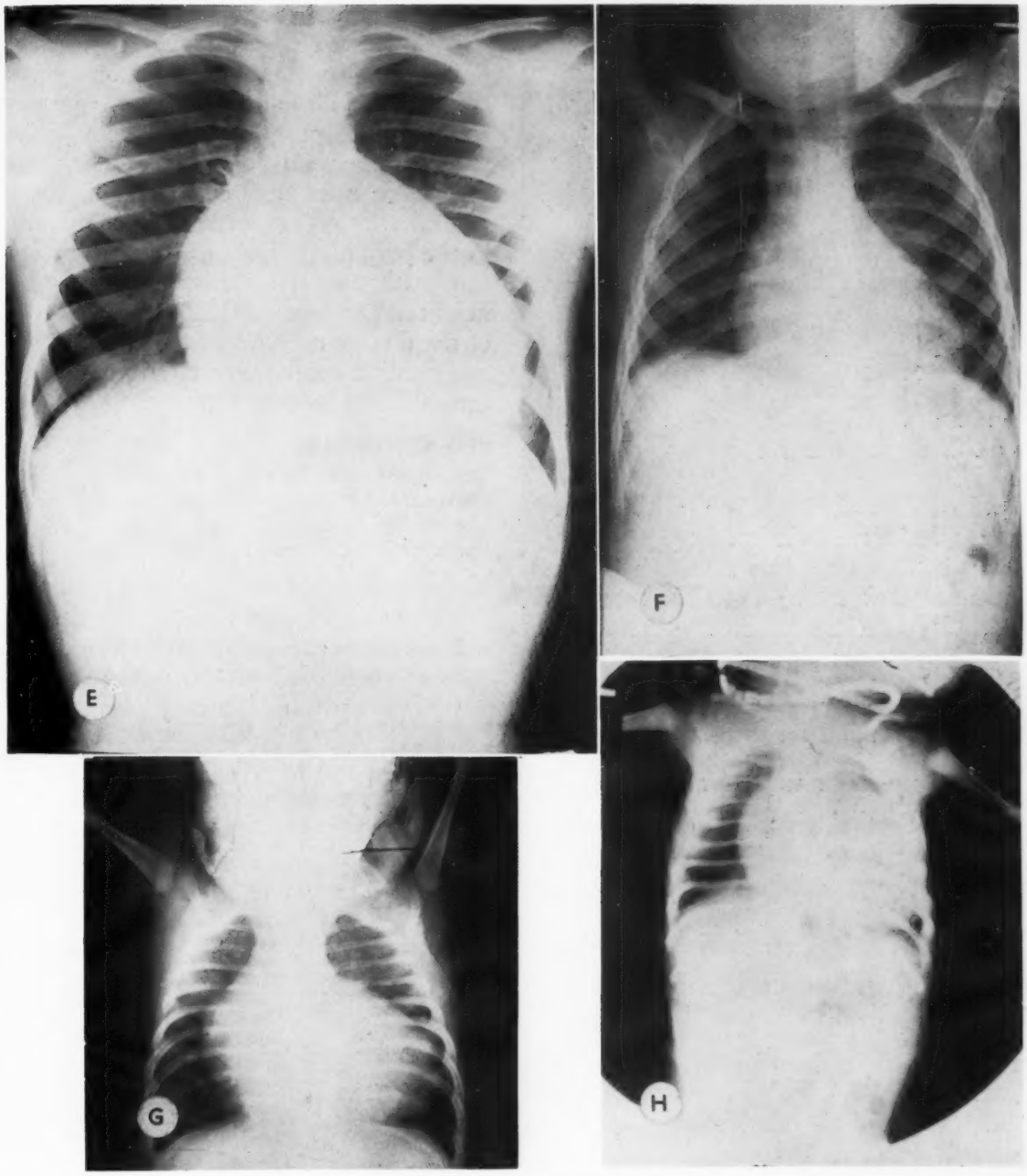

Fig. 4. (*E–H*) Roentgenograms of patients coming to autopsy.

FIG. 5. Lateral roentgenogram. Note the right atrium forming posterior heart border.

raphy has been of little help in the diagnosis of Ebstein's malformation.

SUMMARY AND CONCLUSIONS

1. The pathologic and clinical features of Ebstein's anomaly of the tricuspid valve are briefly reviewed.

2. The roentgen features are correlated with the pathologic findings.

3. A tremendously dilated right heart with normal or decreased pulmonary vasculature and absent left atrial enlargement is the most constant roentgenologic feature in advanced cases. Pulmonary congestive changes and roentgen evidence of pleural effusion are strikingly absent even in the presence of right heart failure.

4. Although the roentgen features are diagnostic in advanced cases, early stages of the disease may present essentially normal cardiac contours or mimic other congenital heart lesions.

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Vol. 81, No. 5

FOREIGN BODIES IN THE HEART*

A REVIEW OF THE LITERATURE SINCE 1952 AND A CASE REPORT

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FOREIGN bodies in the heart have always been of dramatic pathologic interest. We have observed a child with a presumed migratory and non-projectile foreign body imbedded in a defect of the heart itself. A thorough review of the literature of the past five years, since 1952, reveals this to be the only case of its kind reported.

REVIEW OF LITERATURE

Foreign bodies may be divided into four types: (1) projectiles (non-migratory); (2) projectiles (migratory); (3) non-projectiles (non-migratory); and (4) non-projectiles (migratory).

Schinz *et al.*[13] state, "Foreign bodies in the heart almost without exception consist of projectiles." The literature shows this statement to be largely valid, but we also find several instances of non-projectile foreign bodies in the heart.

Migration of foreign bodies within the human body, especially of metallic objects, is well known, and from time to time various case reports of these incidents have been published. Three such reports appeared in a single recent issue of one of the major radiologic journals.[3,9,18] Migration of foreign bodies to the heart occurs more rarely but we find several cases in the literature.

A combination of both factors, or type 4, *i.e.*, a non-projectile foreign body migrating to the heart, is rare indeed. In addition to our case, which has the added factor of a congenital heart defect, we find only 3 other instances cited, 2 of which are somewhat doubtful in that they provide no history of a possible port of entry of the foreign body.

Among those instances of non-migratory foreign bodies of projectile nature, the most common type, Goyette and Keirns[6] and Swan *et al.*[16] describe 4 cases of successful removal of such bodies from the heart. Vickers[19] and Leider and Miscall [10] report 2 cases of bullet wounds in the chest, the bullets being lodged in the heart. The case reported by Vickers was not operated upon and lived until the age of ninety, thirty years after the wound occurred. The other case was successfully operated upon. Boggio Gilot[2] cites the rare case of retention of shrapnel in the myocardial wall for thirty-nine years. The patient had no symptoms referable to the heart, and the shrapnel was discovered by chance in a roentgenogram made when the patient had bronchitis.

Evidence of migration to the heart of foreign bodies of projectile nature is not infrequent. Simpson[14] presents a case with a foreign body in the heart of projectile and embolic nature, which had migrated to the right ventricle from wounds of the lower extremities. Roche and Gravier[12] report the case of a young man who was accidentally wounded by a stray bullet while be was fishing in the Seine River. He was knocked to the ground and was frightened, but had no complaints other than a pain in the right side. Subsequent examination revealed a small wound scar above the right iliac crest, and a bullet in the right ventricle of the heart. The authors present a theory concerning the route of the foreign body, *i.e.*, via the veins of the limbs, the vena cava, and thence to the right ventricle; this

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route is also suggested by Simpson.[14] Dneprovolzhskiy[4] reports a somewhat similar instance and suggests the same pathway of migration. He tells of a twenty-eight year old war veteran who was wounded in the left hip and the soft tissue of the left shoulder. This occurred in 1943, and at that time roentgen examination of the chest and heart was completely negative. A roentgenogram of the soft tissue of the shoulder showed an 8 by 6 mm. metal piece embedded there. In 1945, a roentgenogram demonstrated the metal fragment to be in the right ventricle.

Another case of shrapnel wound, with the metal migrating from under the fifth rib of the right chest to the right ventricle is presented by Stepanova.[15] The wound occurred in 1944. The fragment was successfully removed from the heart in 1951.

We found only 2 or 3 instances in the literature of the past five years of non-projectile foreign bodies which did *not* migrate to the heart; *i.e.*, they were placed directly and purposefully into the heart. This low number, we feel certain, is not indicative of the rarity of this phenomenon, for there must be many unreported cases of stabbings of the heart, mostly resulting in death. Beahrs and Nattox[1] report the successful removal of a needle from the left ventricle of a psychotic woman who had attempted suicide. Tänzer[17] cites another case of a non-migrating, non-projectile foreign body in the heart. He removed the end of a broken cannula from the right ventricle of a man who two years previously had had an intracardial injection. Following removal of the cannula, which was between 6 and 7 cm. long, the patient died. Autopsy revealed that thrombosis had developed along the entire line of the site of the cannula. Karavanov[8] reports the successful removal of a needle from the left ventricle of a woman, in 1954, but there is no history, even conjectural, as to how the needle might have reached the heart; so we cannot say whether it migrated or not.

Of those instances of a non-projectile foreign body migrating to the heart, we have, in addition to our own case, 3 others, one of which presents clear-cut evidence of migration of the foreign body, and the remaining 2 may be presumed to have migrated. Farber and Craig[5] report the case in which a broom straw was found, at autopsy, to be lodged in a thrombus attached to the tricuspid valve in the right atrium of a one month old infant, and Mamedov[11] gives a fascinating case history of the removal of a needle imbedded 4 mm. into the apex of the heart. Mamedov states that a roentgenogram showed the blunt end of the needle to be nearly touching the lung. The patient's complaint was a sharp, stabbing pain in the left chest, but unfortunately, when the presence of the needle was discovered, the patient could supply no information as to its possible port of entry, for he was the victim of amnesia. If the patient is ever cured of his amnesia, his contribution to medical literature could be invaluable.

Jankiewicz[7] offers perhaps the most clear-cut evidence of the migration of a needle into the heart. He firmly believes that such foreign bodies can and do migrate to and from the heart, and after citing several classic instances, some dating back to 1904, he presents his case of a thirty-one year old farmer who was thrown in jail, became depressed, attempted suicide by stabbing himself with a needle, and reported to the hospital twelve days later, having apparently changed his mind about self-destruction. He had made two attempts to get the needle in, the first being unsuccessful. There were in the region of the left nipple two small scars. A roentgenogram was taken which showed a foreign body, apparently a needle, near the pericardium. This was seen only during inspiration. The needle was approximately 2 cm. below the skin. Another roentgenogram a day or so later revealed the needle to be 2.5 cm. below the surface, with the sharp end beginning to enter the pericardium. Decision was made to operate and remove the needle by pulling it by its blunt end from the pericardium. At operation, three

days later, the needle was not visible, though there was an area of the pericardium which was thickened. This thickened area was incised and the blunt end of the needle was seen. It had bored its way 3 cm. into the myocardium.

In our case, the foreign body, a needle, is believed to have migrated to the heart after entry into the body via peripheral soft tissue, or, less likely, through one of the orifices of the body. However, there is no clear-cut history of just where or how the needle entered the body. In this respect the case is similar to the one reported by Mamedov.[11]

REPORT OF A CASE

H. F., a seven year old boy, was admitted to the Shiraz Medical Center, Shiraz, Iran, on March 12, 1956, with a history from a referring physician of the presence of a foreign body in the heart (seen in the roentgenogram of the chest) and also the presence of a congenital heart disease. The history obtained from relatives revealed that the child was well until the age of four years, at which time, following a febrile illness, the child developed generalized edema lasting about two months. This was followed by bouts of recurrent nocturnal dyspnea which persisted until the time of admission.

Physical Examination. Temperature 99.2°F, pulse rate 95, respiration rate 25. Blood pressure 80/60. Height 40 inches; weight 32 pounds. The positive findings were: marked precordial bulge, diffuse precordial systolic thrill, Grade 4 harsh systolic murmur along the left, lower sternal border, and Grade 2 diastolic murmur along the left, upper sternal border. There were also bilateral inguinal hernias and a mild congestion of the pharynx.

Fig. 1.

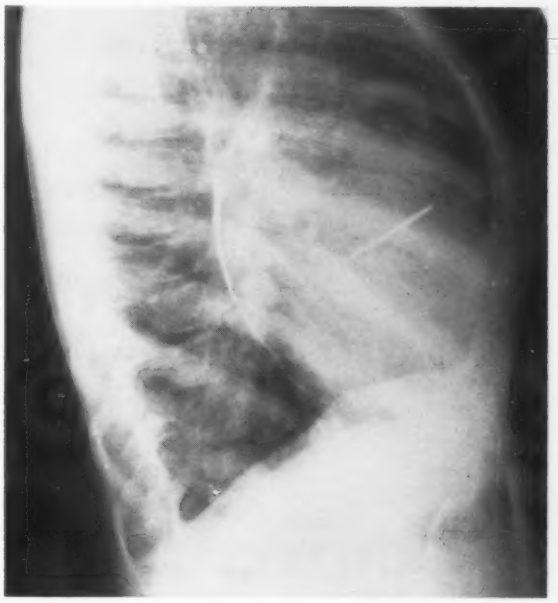

Fig. 2.

Laboratory Findings. Hemoglobin 13.2 gm. per cent, white blood cell count 25,000, with a marked shift of the leukocytes to the left. (This was believed to be due to pharyngitis.) Urinalysis was negative. The stool was negative for ova and parasites. Electrocardiogram suggested combined right and left ventricular hypertrophy with preponderance of the former.

Roentgen Findings. Roentgenograms of the chest in various projections (Fig. 1–4), revealed the presence within the heart of a needle-like metallic object, measuring about 4 cm. in the longest projection. At fluoroscopy, the needle was noted to be fixed on the right posteriorly, and the free end moved rhythmically up and down anteriorly with systolic contraction of the left ventricle. The right ventricle was enlarged and the pulmonary vascular markings somewhat exaggerated. The diagnoses considered were (1) interventricular septal defect, (2) interatrial septal defect. The former was preferred on the basis of clinical findings.

Indications for Cardiac Exploration. It wa-

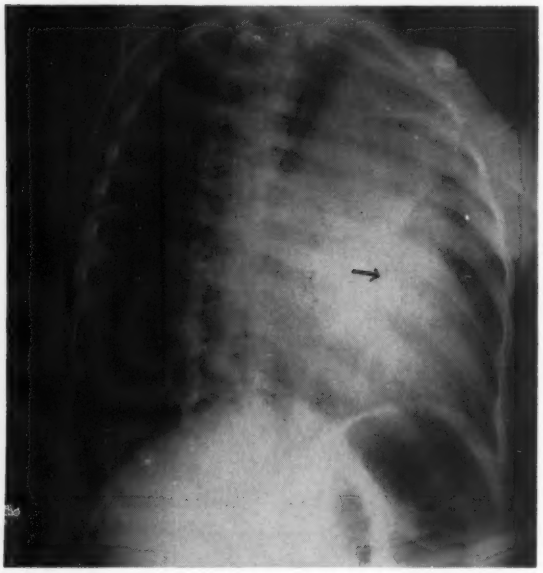

FIG. 3.

our impression that the patient had a congenital heart disease in addition to a foreign body within the heart. It was thought that perhaps the foreign body was aggravating the already diseased heart and possibly serving as a nidus of infection. For this reason, a cardiotomy with the aim of removal of the foreign body was attempted.

Operative Findings. Marked cardiac enlargement, especially of the right ventricle, was noted. The pulmonary artery was also markedly engorged and distended. A needle measuring 4.5 cm. in length was found to be lodged high in the interventricular septum and projecting into the right ventricle. The needle was dislodged from the septum by gentle squeezing of the cardiac apex and was removed by delivery through the pulmonary artery. An attempt was then made to correct the cardiac anomaly which from the configuration of the heart was assumed to be a pulmonary stenosis. Irreversible cardiac arrest ensued. Postmortem examination of the heart revealed a 5 mm., high interventricular, septal defect and marked pulmonary artery dilatation.

DISCUSSION

Several points are raised by our study. In the first place, we find that non-projectile foreign bodies in the heart are more common than one would suppose. In the 18 cases referred to, including our own, there were 11 foreign bodies of projectile nature and 7 of non-projectile nature. Interestingly enough, of the latter group, 5 were needles, the other 2 being a broken cannula and a broom straw. With the exception of the woman who tried to commit suicide by shoving a needle into her heart, the man with a broken cannula in his heart, and Jankiewicz's would-be suicide, we have little evidence as to how the other non-projectile foreign bodies arrived at their cardiac destinations.

Again considering the whole group of 18 cases, we find that we are certain of 9 "non-migrating" cases, so to speak. In the 9 other cases, there is evidence that in 5 the foreign bodies were of embolic nature. These include 4 of projectile type and Jankiewicz's case[7] of the man who stabbed himself with a needle. Whether Karavanov's patient[8] was stabbed in the heart, stabbed elsewhere, swallowed the needle, or tried to commit suicide is not known. In other words, did this needle migrate or not? Karavanov gives only a report of the operation, with no history or presenting complaints. Mamedov's amnesiac[11] is also suspect as an example of a migratory foreign body, though the patient did complain of pains in the left chest which, we may

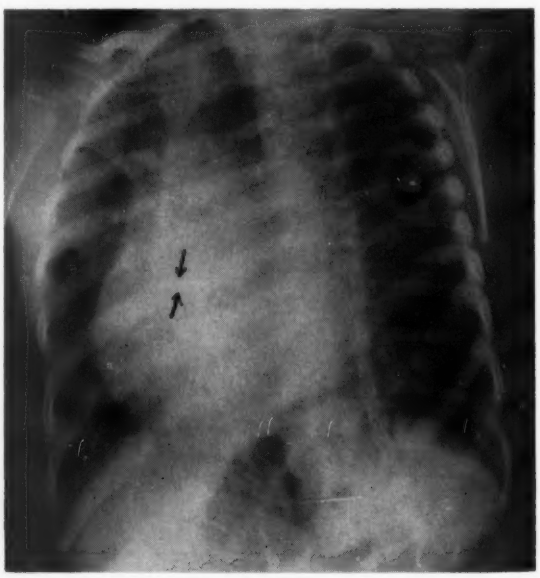

FIG. 4.

surmise, mystified him. The infant reported by Farber and Craig[5] must somehow have swallowed the broom straw, for it is unlikely that it had pierced the skin. Thus, we can be fairly certain of the migratory nature of the foreign body in this instance. In our own case, we feel that the needle in the child's heart was migratory, though we are without conclusive proof. We are convinced, however, that the needle was not put directly into the heart.

Actually, whether a foreign body in the heart is or is not of projectile nature makes little difference. We know that both types can migrate to the heart, and the unanswered question is the route which migrating bodies take. Three authors[4,12,14] suggest the veins of one or another limb, thence to the vena cava and the right ventricle. This seems likely in our case too.

Farber and Craig[5] discuss the fact that foreign bodies frequently invade the heart in cattle. They believe that this is due to gravitation of the heavy ingested material into the second stomach, from which the foreign body bores its way into the precardium and not infrequently into the heart itself. However, this mechanism does not explain similar phenomena in man.

Mamedov[11] believes that his patient's needle entered between the ribs, and through the action of the muscles was forced into the chest cavity. But he emphasizes that this is conjecture only. Jankiewicz[7] feels that far more foreign bodies migrate to the heart by routes other than circulatory than is commonly supposed. He states that foreign bodies of non-projectile nature, such as the needles, are often swallowed or enter through the skin and soft tissue, and by muscle action are propelled toward the heart. To him, the contraction of the heart itself seems to have a "milking" effect which draws a foreign body toward it.

To date, however, there is no proved or universally accepted mechanism which can account for the migration of foreign bodies toward the heart in man. Nor has there been an entirely satisfactory explanation of the routes which foreign bodies choose for their migration to the heart and to the mediastinum. Animal experimentation may shed some light on this question.

SUMMARY

A review of the literature on foreign bodies found within the heart is presented. Foreign bodies in the heart may be either of projectile or non-projectile nature, and may be either migratory or non-migratory.

The case of a child with a presumed migratory and non-projectile foreign body imbedded in a congenital defect of the heart is reported. It is presumed that the foreign body, a needle, migrated to the heart after having been introduced into the body through peripheral soft tissue or through one of the external orifices of the body.

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Vol. 81, No. 5

NORMAL PULSATIONS IN THE PULMONARY VASCULAR TREE AS SEEN WITH ROENTGENOSCOPIC IMAGE AMPLIFICATION*

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PHYSIOLOGIC limitations of the eyes under conditions of conventional roentgenoscopy do not allow recognition of pulsations in the smaller branches of the pulmonary arteries. When the roentgenoscopic image is increased in brightness by image amplification, the pulsations in the pulmonary arteries, particularly the smaller branches, become visible. Criteria for normal pulsations as seen with conventional roentgenoscopy do not apply when image amplification is employed. A normal pulsation in the pulmonary artery branches of the hilar areas might be misinterpreted as "hilar dance" if the difference between the two types of roentgenoscopy were not realized.

Roentgen kymography will demonstrate the pulse waves of the main pulmonary artery segment of the left heart border, but cannot be used to study pulsations in the smaller pulmonary arterial branches within the lung.

A study of pulmonary artery pulsations in relation to pulmonary artery pressure and pulmonary blood flow was begun about three years ago. Careful roentgenoscopic examination with image amplification was carried out in all patients undergoing cardiac catheterization at the Cardio-vascular Laboratory of the Emory University School of Medicine. To date 115 patients have been studied. These data are being analyzed currently and will be reported in the near future.[2]

Because of the above mentioned project, it was felt essential that a normal series of patients be studied as a control group. It is the purpose of this report to present the variations in pulmonary artery pulsations as seen in a group of 100 patients with no demonstrable pulmonary or cardiovascular abnormality by clinical examination.

In frontal projection with conventional roentgenoscopy, lateral movements of the main pulmonary artery segment along the left heart border can be easily identified as can the movement of the left ventricle and aortic arch. When one observes the left pulmonary artery branch in the left hilar area, pulsations are only faintly visible, and, as one follows these branches peripherally, their expansile pulsation is rapidly lost to visualization as the segmental arteries arise. The right descending pulmonary artery in the right hilus in the most normal subjects cannot be seen to pulsate when viewed with conventional roentgenoscopy. No pulsatile activity is evident in the basilar arterial branches on either side by conventional roentgenoscopy.

When roentgenoscopic image amplification is used, the visible pulsations within the segmental branches of the pulmonary artery become easily visible. With each systole the secondary arteries in the lung fields can be seen to expand and show an increase in density as blood flows through the vessels.

For purposes of description, the following grading system of pulsations was devised:

- 0 = no perceptible pulsation with image amplification.
- + = slight pulsations easily visualized with image amplification (average normal).
- ++ = more prominent pulsations, borderline between upper limits of normal and abnormally increased.
- +++ = definite and marked accentuation of pulsation over average normal.

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It is realized that the data to be presented are based entirely upon subjective impressions, there being no way to obtain objective evaluation except by cineroentgenography. Even with cineroentgenography the impression as to the degree of pulsation present is subjective.

The first 24 subjects were examined in upright and horizontal positions, before and after exercise. Since position was shown to have no effect on the pulmonary artery pulsations, the remaining 76 subjects were examined in supine position. Moderate to severe exercise was shown to increase the pulmonary artery pulsations only slightly in the periphery of the lung, though there was a moderate increase in the prominence of the pulsations of the main and hilar branches of the pulmonary artery. Since patients are normally examined in a resting state and since a better base line for normal variation was obtained when patients were examined at rest, observations after exercise were omitted in the last 76 subjects.

Observations of pulsations in the pulmonary arteries were specifically recorded at five main points (Fig. 1): (1) The main pulmonary artery segment of the left heart border; (2) the left pulmonary artery branch as it descends through the left hilus; (3) the right descending pulmonary artery branch in the right hilus; (4) the peripheral segmental branches of the left pulmonary artery in the basilar divisions of the left lower lobe; and (5) the peripheral segmental branches of the right pulmonary artery in the basilar divisions of the right lower lobe.

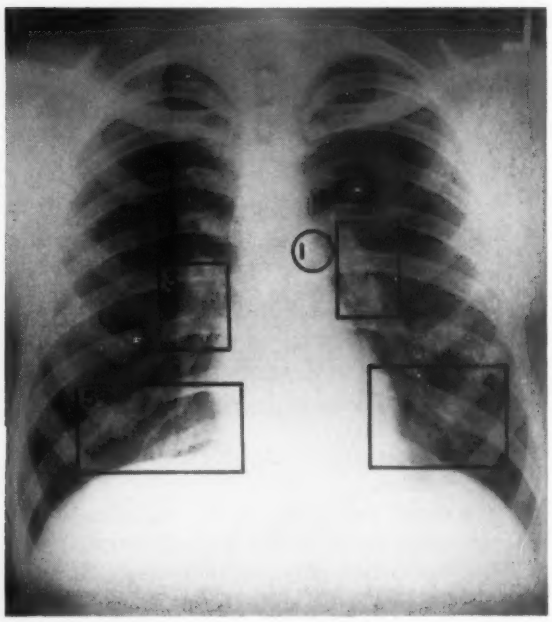

Fig. 1. Pulsations as observed with roentgenoscopic image amplification were graded at sites 1 through 5 as indicated on this posteroanterior roentgenogram of the chest.

Arterial Branches of the Right Lung

The right main pulmonary artery is hidden in the mediastinal shadow (Fig. 2). The anterior trunk, the first major branch, is given off within the mediastinum and contributes very little to the hilar shadow; this branch supplies blood to the greater portion of the right upper lobe.[4] The main pulmonary artery continues as the right descending branch and forms the major vascular shadow of the right hilus. This arterial branch bears a constant relationship to the bronchus intermedius which lies medially; the lateral border of the right descending pulmonary artery is demarcated by the lung parenchyma. The greatest transverse diameter of the vessel can be measured accurately on most routine chest roentgenograms and, where the borders are not definite, this branch can be visualized by laminagraphy. In this series, the normal transverse measurement for adults varied between 9–15 mm. Abrams[1] and Schwedel *et al.*[3] have reported the same findings. In a series of 70 chest roentgenograms of normal patients from eight weeks of age to sixteen years of age, the following range of normal variations in the size of the right descending pulmonary artery branch was noted: (1) Infant = 5–8 mm.; (2) six months to ten years = 6–11 mm.; and (3) eleven years to sixteen years = 8–14 mm.

Roentgenograms for measurement purposes were made at a target-film distance of 6 feet during inspiration, and in posteroanterior projection. Roentgenograms of newborn infants were made with the patient in supine, anteroposterior position.

Pulsations of the right descending pulmonary artery branch are always easily visible in normal patients at rest during roentgenoscopy with image amplification. The amplitude of the pulsation is slightly variable. Pulsations of this branch form a baseline for comparison with other branches of the pulmonary artery in both lungs. This vessel can be seen to expand in all directions with each cardiac systole; the vessel shows an associated increase in density during systole. With only a rare exception, pulsations in the right descending pulmonary artery branch are graded 1+ in normal subjects. Exercise increases the prominence of the visible pulsation in the right descending pulmonary artery branch only slightly.

The right descending pulmonary artery branch gives off a small branch to the posterior segment of the right upper lobe in the more superior portion of the lung hilus and proceeds inferiorly and slightly laterally to divide into the right middle and lower lobe arteries, which subdivide according to the bronchopulmonary segments.

As one follows the right descending pulmonary artery branch inferiorly into the segmental arterial trunks to the middle and lower lobes, pulsations become more difficult to visualize. In the normal subject at rest, pulsations in the basilar arteries of the right lower lobe are not usually visible, even though these branches (which average 2 to 4 mm. in size in the adult and 1 to 2 mm. in the child) can be seen with roentgenoscopic amplification. Well defined expansile pulsations in the right basilar arterial trunks with patient at rest are abnormal and when present denote an increase in pulmonary blood flow.[2]

ARTERIAL BRANCHES TO THE LEFT LUNG

The left pulmonary artery is continuous with the main trunk and forms a portion of the pulmonary artery segment of the left heart border (called "pulmonary conus" in older literature). Since there is great variability in the position of the left pulmonary artery branch as it passes through the hilar area, there are no constant landmarks by which this artery can be measured accurately. The left pulmonary artery gives off the major upper lobe branch at the superior border of the hilar shadow and then courses obliquely downward giving off branches to the lingular segment of the upper lobe and to various segments of the lower lobe (Fig. 2).

Fig. 2. Normal pulmonary arterial anatomy as outlined by angiocardiogram.

Pulsations in the left pulmonary artery branch in the hilar area are best seen with the patient supine and rotated about 15 degrees to a right posterior oblique position in relation to the roentgenographic table. Pulsations in the left pulmonary artery are usually more prominent than those seen in the right descending pulmonary artery branch. Sometimes the difference in pulsation of the two arteries is very striking. This normal differential pulsation becomes greatly magnified in the presence of isolated valvular pulmonary stenosis. In this condition, pulsations of the right descending pulmonary artery become hypoactive; pulsations of the left descending branch become hyperactive. Pulsations of the left pulmonary artery branch in the hilar area are graded 1+ or 2+ in normal subjects.

The branches of the left pulmonary artery in the basilar segments of the left lower lobe are also more easily visualized than the same branches in the right lung. The more medial basilar branches (anteromedial basilar artery and posterior basilar artery) are more readily visualized than the more lateral branches. Pulsations in the basilar arterial branches of the left lower lobe are graded 0 to 2+ in normal subjects, the average being 1+.

Pulsations in the branches of the upper lobe segments of both lungs are poorly defined even with roentgenoscopic image amplification. Roentgenoscopic study of these branches has not been found to be of any value in assessing pulmonary blood flow. Also, the pulsations of the main pulmonary segment of the left heart border are quite variable and are not considered important in the evaluation of physiologic changes in the pulmonary circulation.

Pulsations in the smaller basilar branches of the pulmonary arteries in either lung can be accentuated by exercise. The amplitude of these pulsations, however, will never be as great as that in patients having abnormally increased pulmonary blood flow as with left to right shunts. Pulsations of the pulmonary artery basilar branches are more easily visualized during expiration than during inspiration. Frequently no pulsatile activity is seen in these smaller branches during inspiration, whereas the vessel becomes pulsatile as the patient exhales. The Valsalva maneuver produces initially a decreased prominence of pulsations in the small basilar arterial branches, but in time pulsations will tend to return to former prominence. The Müller maneuver accentuates the visible pulsation in the smaller branches of the pulmonary artery.

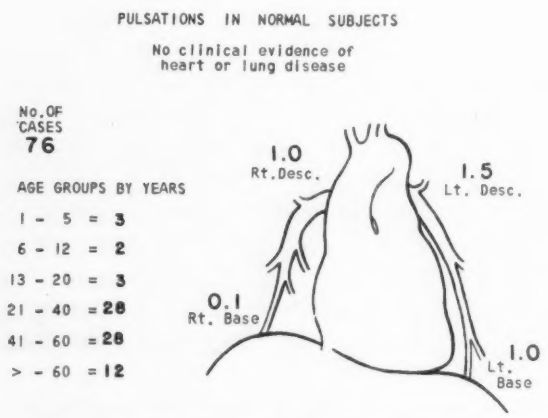

Fig. 3. Pulsations in 76 normal subjects.

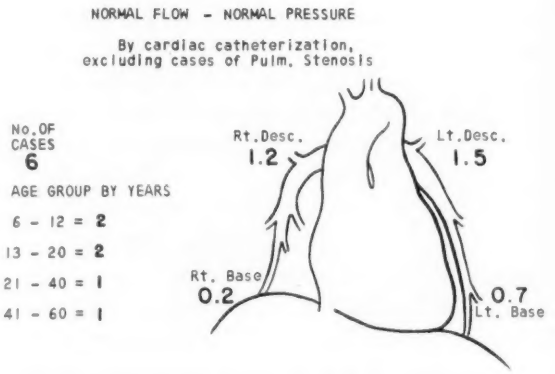

Fig. 4. Pulsations in 6 patients who had cardiac catheterization, with normal blood flow and pulmonary artery pressure and with no evidence of pulmonary stenosis.

Pulsations of the segmental pulmonary arterial branches become obscured in patients with passive congestion and edema due to mitral stenosis or left heart failure since the borders of the vessels are blurred by the presence of transudate in the perivascular lymphatics and interstitial tissues and alveoli of the lung. Roentgenoscopy with image amplification is thus helpful in differentiating the effects of heart failure from those of increased pulmonary blood flow.

In 76 of the normal subjects, the degree of pulsation of central and peripheral arterial branches was averaged. These data are summarized in Figure 3. The numbers adjacent to the central and peripheral arterial branches in the diagram represent the average grading of pulsations at these locations. Similar data are presented in Figure 4 for 6 patients who had cardiac catheterization, with normal pulmonary blood flow and pulmonary artery pressure and with no evidence of pulmonary stenosis.

THE PULMONARY VEINS

The normal pulmonary veins produce less prominent shadows in the lung fields than do the arterial branches. Tributaries of the superior pulmonary veins cross obliquely through the hilar areas (Fig. 5) and are difficult to identify in the normal subject except by laminagraphy or angiocardiography. The superior pulmonary veins contribute very little to the hilar shadows normally, but become very prominent in mitral stenosis, left ventricular failure, and increased pulmonary blood flow with congenital heart lesions.[5] The inferior pulmonary veins are more readily identified on routine roentgenograms of the chest. Three or four secondary tributaries of the right inferior pulmonary vein usually can be identified passing into the right heart silhouette, fusing with the shadow of the left atrium. The normal right border of the left atrium can be visualized through the heart silhouette in about 20 per cent of patients with roentgenoscopic image amplification. In this series, the secondary tributaries of the right inferior pulmonary vein varied from 2–11 mm. in the greatest transverse diameter at the right heart border. These tributaries of the right inferior pulmonary vein can be visualized with roentgenoscopic image amplification and cannot be seen to pulsate. The tributaries of the left inferior pulmonary vein are less easily identified because these are hidden by the heart silhouette.

Fig. 5. Normal pulmonary venous anatomy as outlined by angiocardiogram.

DISCUSSION

Roentgenoscopy with image amplification greatly enhances the study of physiologic changes within the lesser circulation. More complete information concerning the pulmonary circulation can be obtained if one not only employs roentgenograms of the chest, but also takes into consideration the changes in pulsatile activity of the hilar and segmental arterial pulmonary branches within the lungs as seen with roentgenoscopic image amplification. The presence of normal pulmonary blood flow and pressure, increased pulmonary blood flow, decreased pulmonary blood flow, and pulmonary hypertension can be predicted with reasonable accuracy by roentgenographic and roentgenoscopic studies. These observations will be described in a future publication.[2]

SUMMARY

Pulsations of the pulmonary vascular tree are well visualized when roentgenoscopic image amplification is used. Observations in 100 normal subjects are reported. Easily visible expansile pulsations are seen in the right descending pulmonary artery branch in normal subjects. Pulsations at the right pulmonary base are either imperceptible or only faintly visible. The pulsations of the left pulmonary artery are usually more prominent normally than those of the right descending pulmonary artery branch. Also, the left basilar arterial branches are more easily observed than those of the right lung base.

Roentgenoscopic studies using image amplification in patients with increased or

decreased pulmonary blood flow or with pulmonary hypertension are in progress and will be reported in the near future.

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Vol. 81, No. 5

GREAT VESSEL INVOLVEMENT IN LUNG CANCER: ANGIOCARDIOGRAPHIC REPORT ON 250 CONSECUTIVE PROVED CASES*

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ANGIOCARDIOGRAPHY has revealed significant involvement of the great blood vessels in cancer of the lung, chiefly in the superior vena caval and pulmonary arterial systems. Earlier publications have reviewed the findings in the first 100 consecutive proved cases.[5,6,14,15] This report summarizes the data from the angiocardiographic study of 150 additional cases.

METHOD

All the patients included in this study had microscopically proved cancer of the lung. Angiocardiography was performed according to the method of Robb and Steinberg.[9] Premedication and anesthesia were not necessary and all studies were made during inspiration in the erect, sitting position. In order to study the superior vena caval and pulmonary circulatory systems to advantage, the frontal projection was used routinely.[6] At first, large 14×17 inch films with grid cassettes mounted in a standard stereocassette unit permitted two films per injection. Later, after the 12×12 inch roll film magazine became available, serial studies, usually at one second intervals, were also used[16] especially when involvement of the superior vena caval system was suspected. Usually, a second injection was made immediately after processing and inspection of the frontal studies. If the superior vena cava was affected, studies in the lateral projection were made. Right or left anterior oblique views for the homolateral artery were then secured for study of the major pulmonary arteries and their branches. Sodium urokon (70 per cent) in 50 cc. doses was the contrast agent used in the majority of patients; no fatalities or troublesome reactions occurred despite the double injection at short intervals.

As in the previous studies of cancer of the lung,[5,15] the angiocardiograms, conventional roentgenograms, clinical, surgical and pathologic data were reviewed in order to fully evaluate the ravages of the disease. Again, it is desirable to classify the angiocardiographic changes into five groups: (1) those in which no pulmonary vascular change is seen; (2) superior vena caval involvement; (3) stenosis or occlusion of the right or left pulmonary artery; (4) lobar and segmental pulmonary arterial involvement; and (5) pericardial involvement (Table 1).

RESULTS

In 27 of the new series of 150 cases of lung cancer, a peripheral lesion produced no demonstrable change in the segmental branches of the pulmonary artery (Fig. 1). The superior vena caval system was involved in 14 instances (Fig. 2 and 3). The major pulmonary arteries (Fig. 4 to 6) were occluded or distorted in 21 cases, while abnormalities of the lobar and segmental pulmonary arteries (Fig. 7 and 8) occurred in 87 patients (Table 1). Only one patient had demonstrable pericardial extension of cancer (Fig. 9). Twenty of the patients were women, an incidence of 8 per cent. The ages varied between thirty-one and eighty years.

Involvement of the superior vena caval system in primary cancer of the lung varied from polypoid invasion of the superior vena cava (Fig. 2, *A*, *B*, *C* and *D*) to partial and complete occlusion of the superior vena cava by adjacent masses (Fig. 3, *A*, *B* and

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TABLE I

VASCULAR CHANGES REVEALED BY ANGIOCARDIOGRAPHY IN LUNG CANCER
(CASES CLASSIFIED ACCORDING TO PRINCIPAL LESION)

	First 100 Cases[15]	Next 150 Cases	Per Cent of 250 Cases
1. No pulmonary vascular changes with			
(a) no parenchymal disease on roentgenography	2	3	
(b) peripheral lung cancer	8	16	
(c) peripheral lung cancer with pleural fluid	1	3	
(d) hilar or mediastinal cancer	2	5	
Total	13	27	16
2. Involvement of superior vena cava system			
axillary vein	2	1	
subclavian vein	4	2	
innominate vein	2	1	
superior vena cava	9	10	
Total	17	14	12
3. Major pulmonary artery involvement			
right	4	4	
left	21	17	
Total	25	21	18
4. Lobar and segmental pulmonary artery involvement	44	87	53.2
5. Pericardial invasion	1	1	0.8
All Cases	100	150	100.0

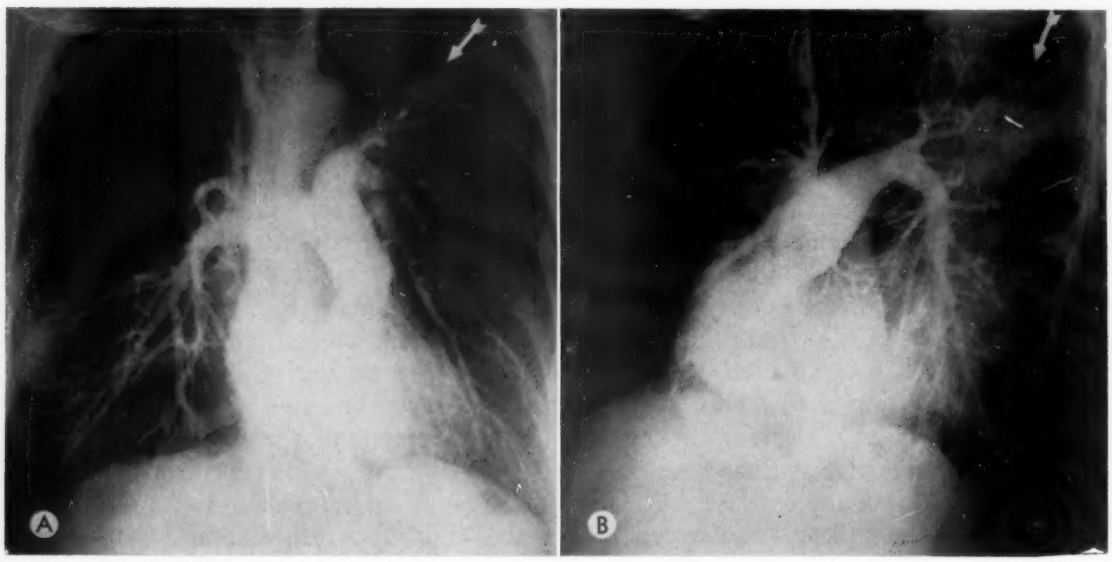

FIG. 1. *Intact pulmonary arterial circulation in a fifty-two year old asymptomatic woman with peripheral lung cancer, treated by left upper lobectomy.* (*A*) Frontal (14×17 inch) teleangiocardiogram shows a normal pulmonary arterial system. Arrow points to left upper lobe cancer. (*B*) Left anterior oblique (14×17 inch) teleangiocardiogram reveals normal pulmonary artery circulation in the posterior segment of the left upper lobe (arrow).

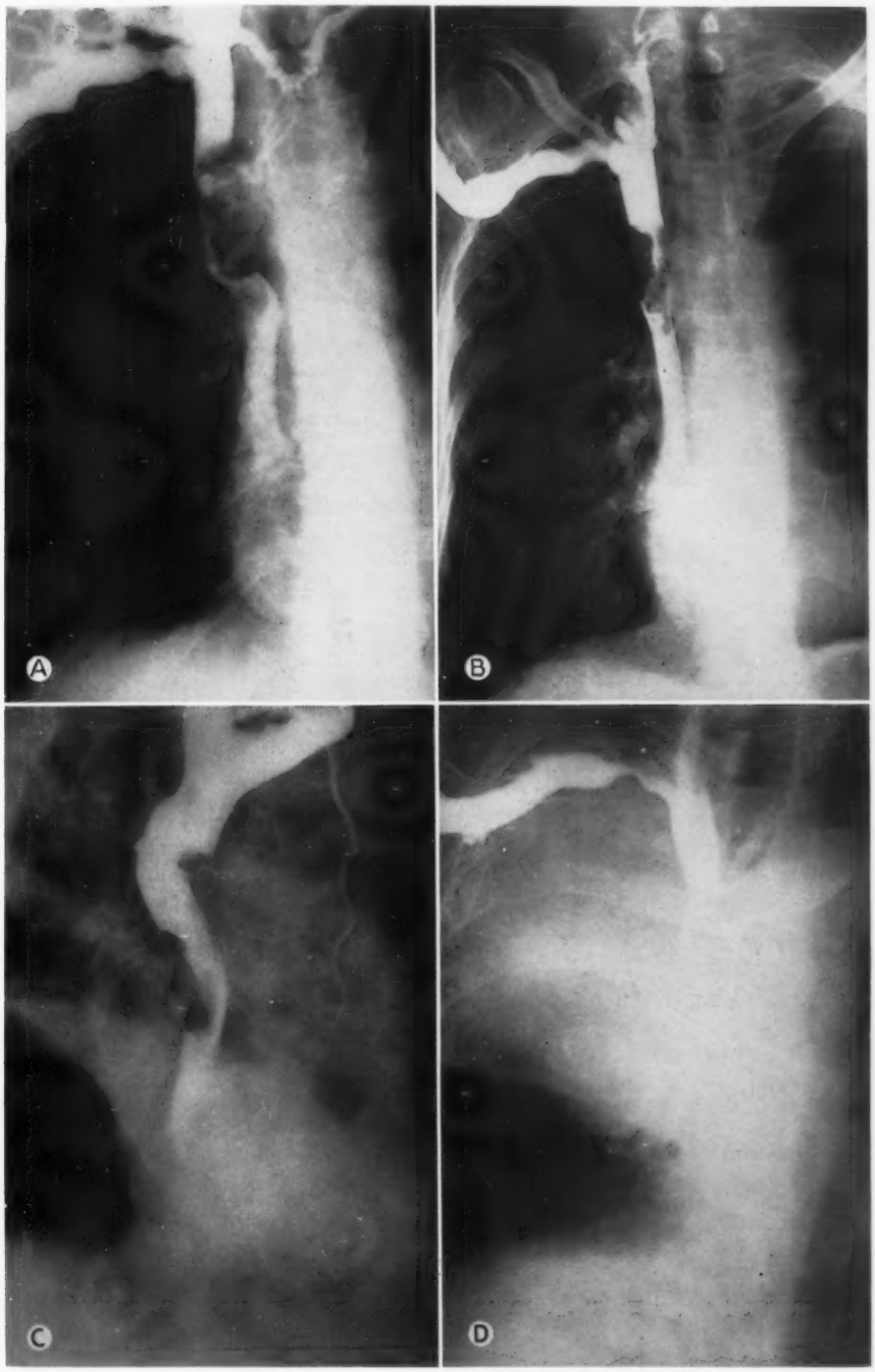

FIG. 2. *Polypoid invasion of the superior vena cava by lung cancer.* (*A*) Frontal angiocardiogram (serial, 12×12 inch study) of a forty-three year old woman with superior vena caval syndrome. There is prominent polypoid invasion of the superior vena cava. Treatment consisted of sternal decompression and roentgen therapy with temporary improvement. (*B*) Frontal teleangiocardiogram of a fifty-three year old man with superior vena caval syndrome. Temporary improvement followed sternal decompression and radiation therapy, but death ensued within a year. (*C*) Serial right anterior oblique angiocardiogram showing polypoid invasion of the superior vena cava in a seventy year old man. Death occurred five weeks later despite roentgen therapy. (*D*) Frontal teleangiocardiogram of a fifty-four year old man with extensive right upper lobe cancer and polypoid invasion of the superior vena cava.

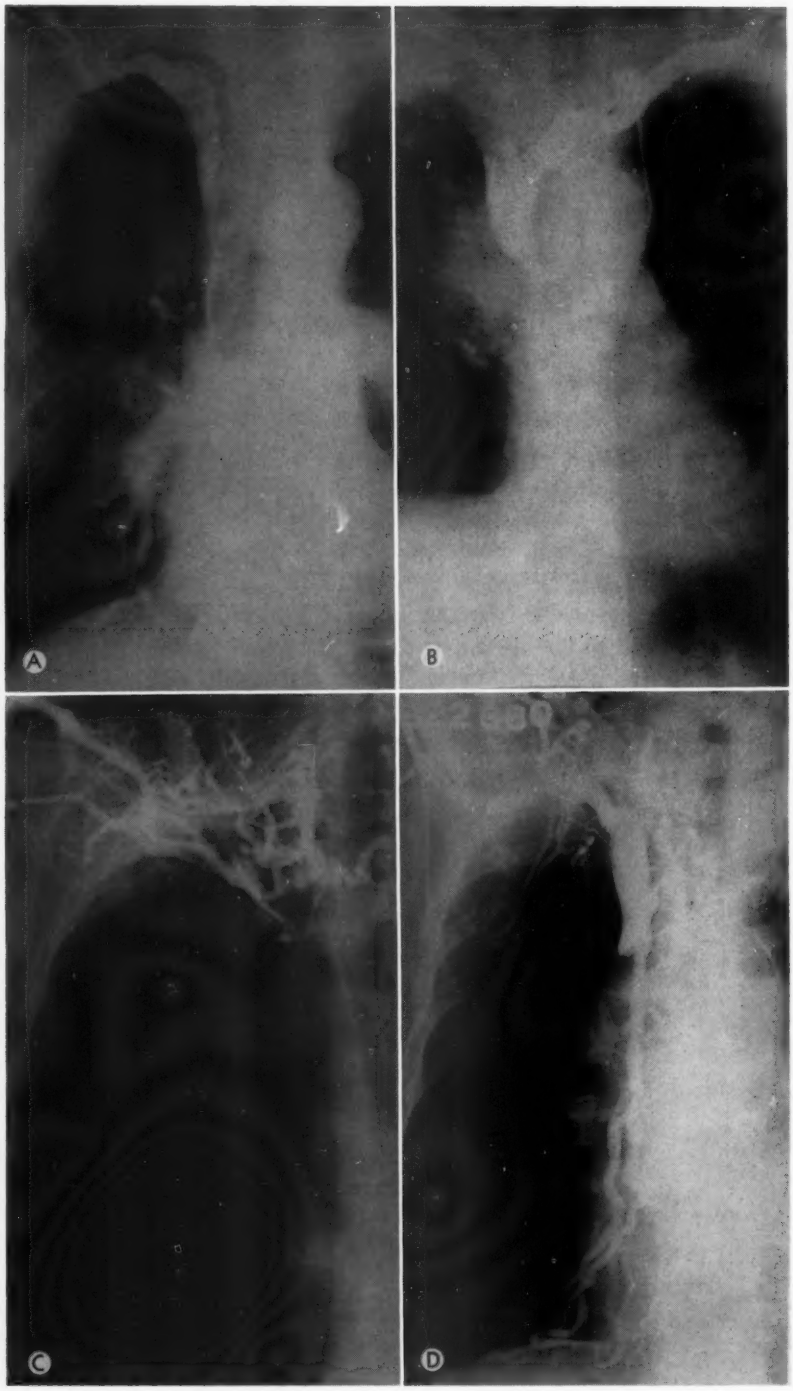

FIG. 3. *Stenosis and occlusion of the superior vena caval system by lung cancer.* (*A*) Frontal teleangiocardiogram reveals marked constriction of the superior vena cava by mediastinal spread of lung cancer in a sixty-nine year old man. Confirmed by exploratory thoracotomy; patient died four months later. (*B*) Frontal teleangiocardiogram shows superior vena caval obstruction by a large mediastinal-hilar mass in a sixty-two year old man. Exploratory thoracotomy confirmed inoperability; roentgen therapy gave temporary relief; death followed in nine months. (*C*) Frontal teleroentgenogram demonstrates complete occlusion of the right innominate vein in a forty-two year old man, also confirmed by exploratory thoracotomy. (*D*) Frontal teleroentgenogram shows complete obstruction of superior vena cava in a forty-nine year old man with collateral circulation due to mediastinal-hilar spread of lung cancer; patient died nine months later despite roentgen therapy.

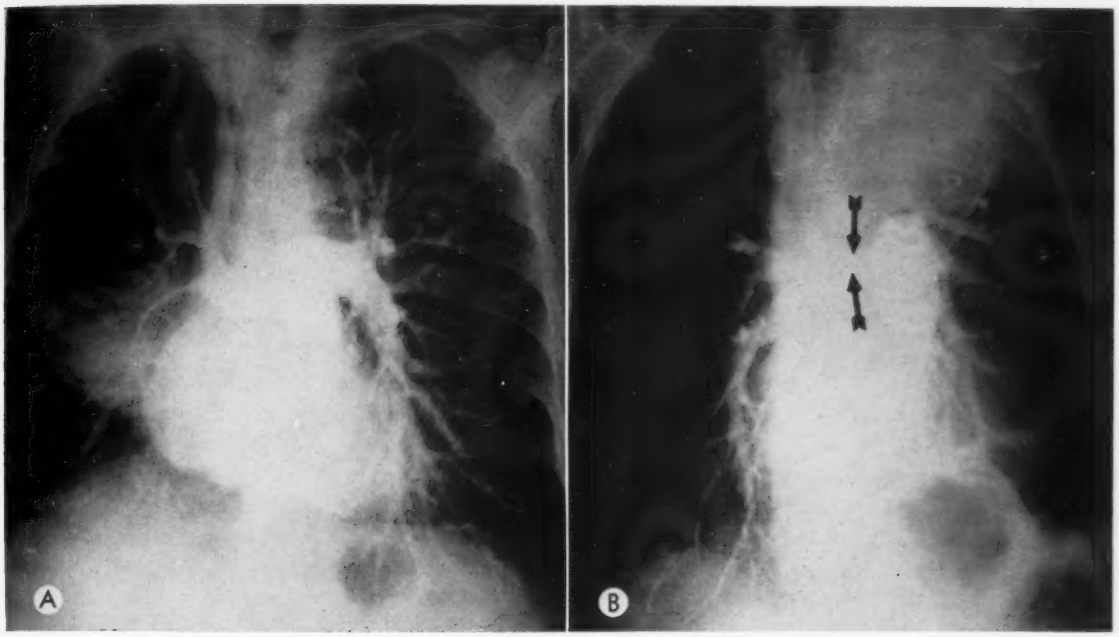

Fig. 4. *Major right pulmonary arterial invasion by lung cancer.* (*A*) Frontal teleangiocardiogram shows invasion of the descending branch of the right pulmonary artery in a fifty-nine year old man. Exploratory operation revealed a nonresectable 15 cm. tumor invading pericardium. (*B*) Frontal teleangiocardiogram discloses cancer of the left upper lobe causing distortion of the segmental pulmonary arteries. More important in determining inoperability is the partial constriction by left hilar cancer (arrows) of the right pulmonary artery at its origin.

D). Complete occlusion of the right innominate vein is demonstrated in Figure 3*C*. Occlusion of the right pulmonary artery proximal to the bifurcation into superior and inferior branches is shown in Figure 4*A*. Figure 4*B* demonstrates cancer of the left upper lobe extending into the hilus and constricting the opposite right pulmonary artery at its origin. Partial involvement of the left pulmonary artery, usually caused by large hilar masses, is shown in Figure 5, *A*, *B*, *C* and *D*. Complete occlusion of the left pulmonary artery by left hilar masses is seen in Figure 6, *A*, *B*, *C* and *D*. Cancer of the right lung with involvement of the right pulmonary artery beyond its bifurcation at the hilus is illustrated in Figure 7*A*. Involvement of the left lung without obstruction or occlusion of the left pulmonary artery at the hilus is revealed in Figure 7, *B* and *C;* these findings suggest that pneumonectomy is feasible. Figure 8*A* shows atelectasis of the right upper lobe with resulting distortion of the pulmonary arterial circulation. Figure 8*B* demonstrates complete occlusion of the left upper lobe artery by cancer of the left upper lobe. Finally, right atrial, pericardial, pulmonary venous and left atrial invasion by cancer of the right lung is seen in Figure 9, *A* and *B*. Increased vascularity of lung cancers at the time of systemic blood flow (the so-called "tumor staining") is shown in Figure 10, *B* and *D*. The abnormal tumor circulation arises from the bronchial arteries.

DISCUSSION

The value of angiocardiography for determining resectability of lung cancer when there is serious involvement of the great vessels has been amply confirmed.[1,2,4,11,12,13,17] The most striking vascular deformities in lung cancer occur when there is hilar and mediastinal involvement (Fig. 2 to 9). In a series of 250 consecutive cases, 30 per cent had superior vena caval and major pulmonary artery disease (Table 1). The re-

maining 70 per cent were chiefly patients with peripheral lung cancer. Here angiocardiography, by demonstrating lack of serious involvement, provided preoperative evaluation of the great vessels. Such lesions, in keeping with the recent views of the Mayo group,[3] should be considered operable. However, resectability cannot always be clearly determined. For instance, in a patient with right lung cancer (Fig. 7*A*), the right pulmonary artery was uninvolved at the hilus, but exploration revealed wide-

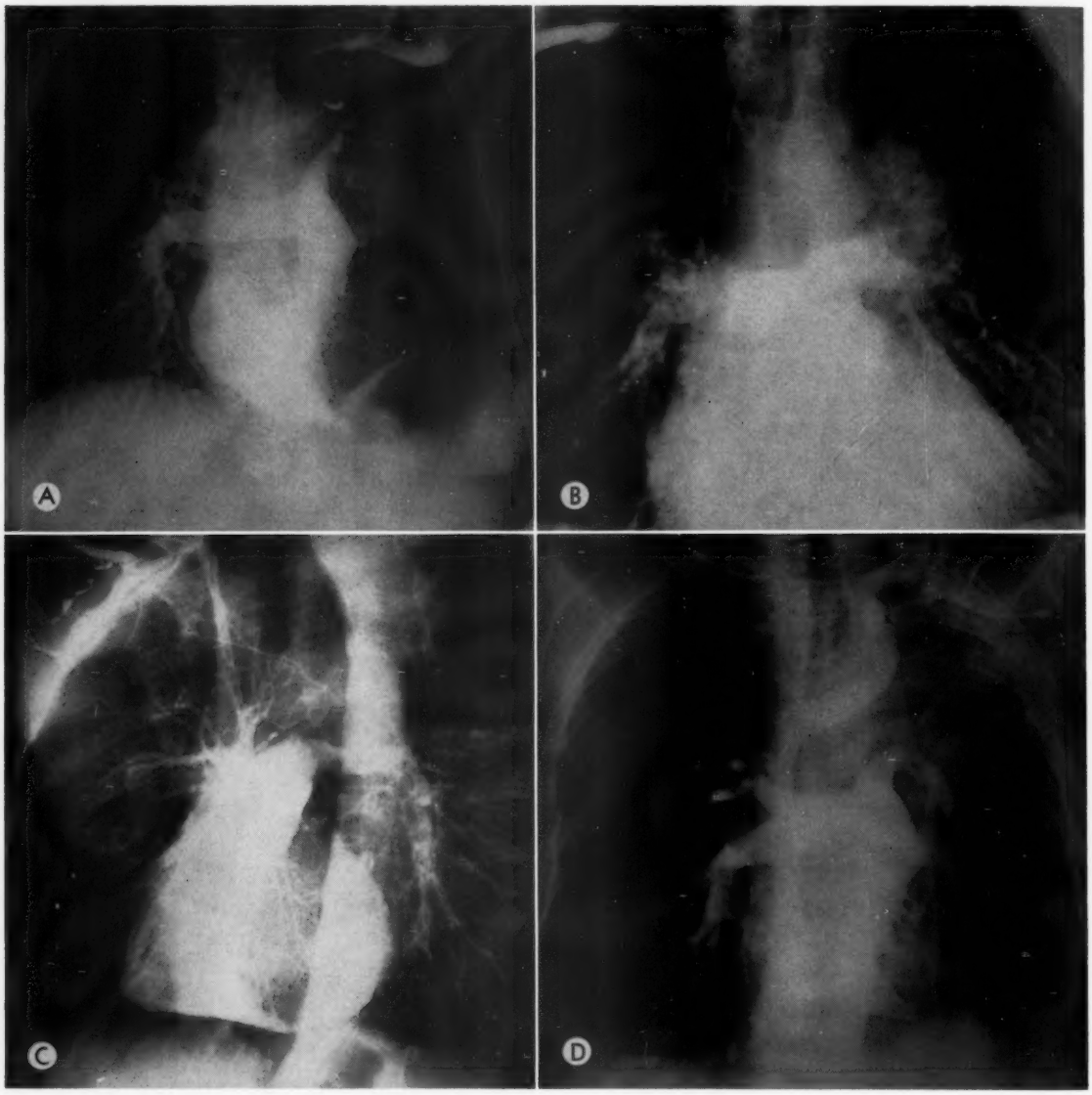

FIG. 5. *Partial obstruction of the left pulmonary artery by lung cancer.* (*A*) Serial frontal angiocardiogram of a fifty-one year old woman shows almost complete occlusion of the left pulmonary artery (only the left upper lobe circulation is spared). (*B*) Serial frontal angiocardiogram of a sixty-five year old man reveals invasion of the left upper lobe pulmonary arterial circulation by a hilar mass; exploratory thoracotomy revealed a nonresectable left hilar mass invading the pericardium. (*C*) Left anterior oblique teleangiocardiogram discloses constriction of the left pulmonary artery by a left superior mediastinal mass. Lobectomy was performed despite invasion of the chest wall and adherence to the great vessels. Death in six months followed empyema and erosion of the left subclavian artery. (*D*) Frontal teleangiocardiogram reveals marked kinking of the left pulmonary artery by mediastinal-hilar cancer in a sixty-three year old man. Operation was not performed because of development of recurrent nerve paralysis.

spread nonresectable disease. On the other hand, despite widespread disease of the left lung in another patient (Fig. 7, *B* and *C*), the left pulmonary artery proved to be intact and permitted pneumonectomy. Figure 8, *A* and *B* reveals that only the right and left upper lobes were involved respectively in 2 other patients. However, operation in the patient with disease of the left lung revealed unresectable cancer. As illustrated in Figure 9, *A* and *B*, advanced extension of cancer to involve vital cardiovascular structures such as the superior vena cava, right atrium, pulmonary veins

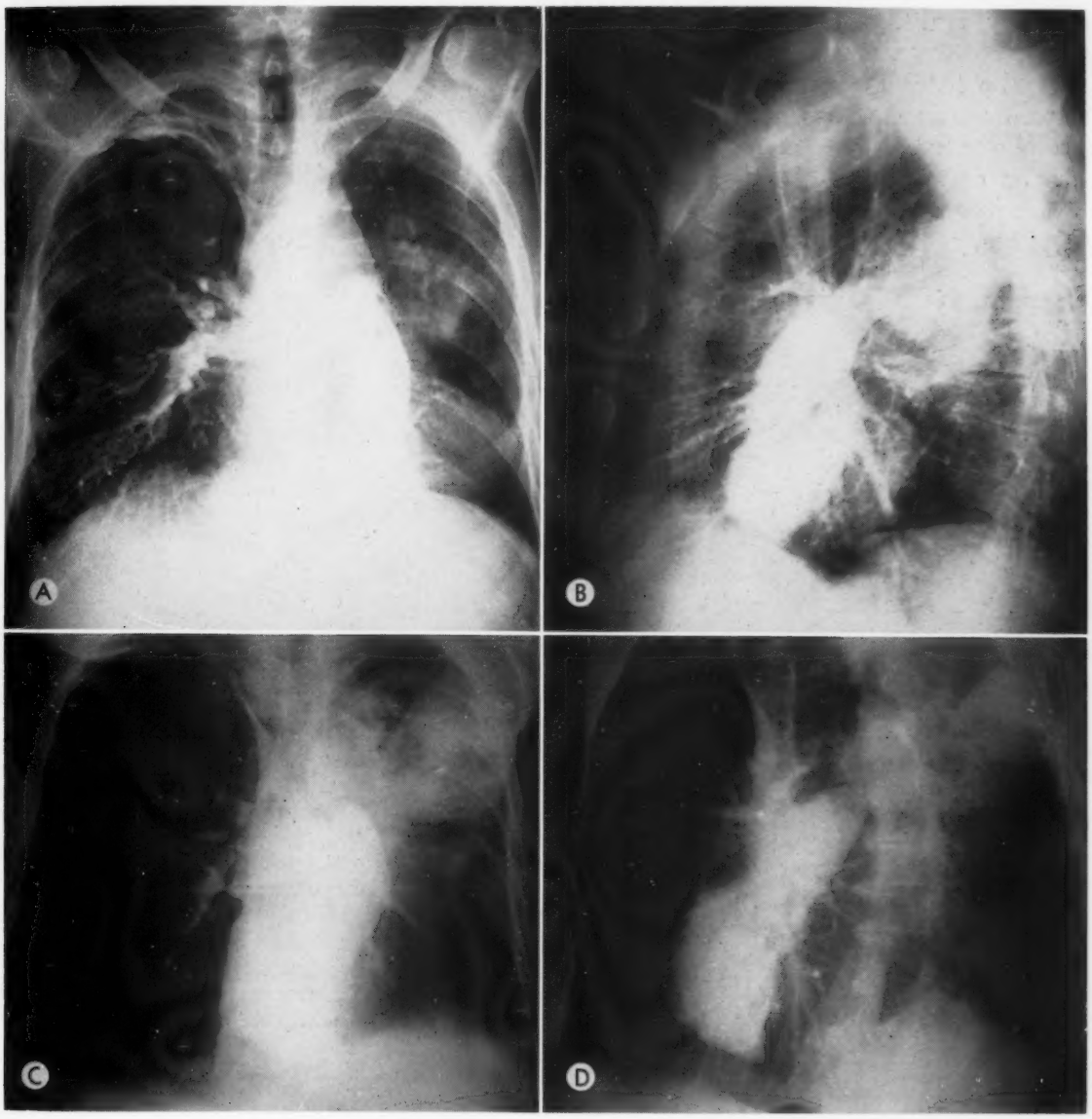

Fig. 6. *Complete obstruction of the left pulmonary artery by lung cancer.* (*A*) Frontal teleangiocardiogram shows complete obstruction of the left pulmonary artery in a fifty-three year old man. (*B*) Left anterior oblique teleangiocardiogram of the same patient also reveals absence of visualization of the left pulmonary artery; exploratory operation disclosed a "frozen" nonresectable hilus due to cancer. (*C*) Frontal teleangiocardiogram shows complete absence of visualization of the left pulmonary artery in a sixty-six year old man. (*D*) Left anterior oblique teleangiocardiogram of the same patient confirms the complete occlusion of the left pulmonary artery.

and left atrium may also be demonstrated.

Angiocardiography, by itself, cannot be depended upon to establish the diagnosis of primary cancer of the lung. This is because disease of the neighboring mediastinal structures may produce identical defects. For instance, superior vena caval obstruction can be caused by malignant primary and metastatic mediastinal tumors, aortic aneurysms, mediastinitis and thromboses.[6,10] Polypoid invasions of the superior vena cava can also be caused by lymphomata.[5,6] Complete and partial occlusion of the main branches of the pulmonary artery, while strongly presumptive of primary lung cancer, may also rarely be caused by other diseases. Distortion and diminished vascularity of the pulmonary arterial tree, contrary to earlier reports,[7,8] should not be relied upon for diagnosis of bronchial cancer. Only when histologic proof of primary lung cancer is available should cardiovascular changes be attributed to cancer.

When carcinoma of the lung is complicated by severe chronic pulmonary disease such as pulmonary fibrosis and emphysema, angiocardiographic study may, as illustrated in Figure 11, *A*, *B* and *C*, disclose that the hazard of pneumonectomy is too great because of the burden which would fall on the diseased opposite lung. However,

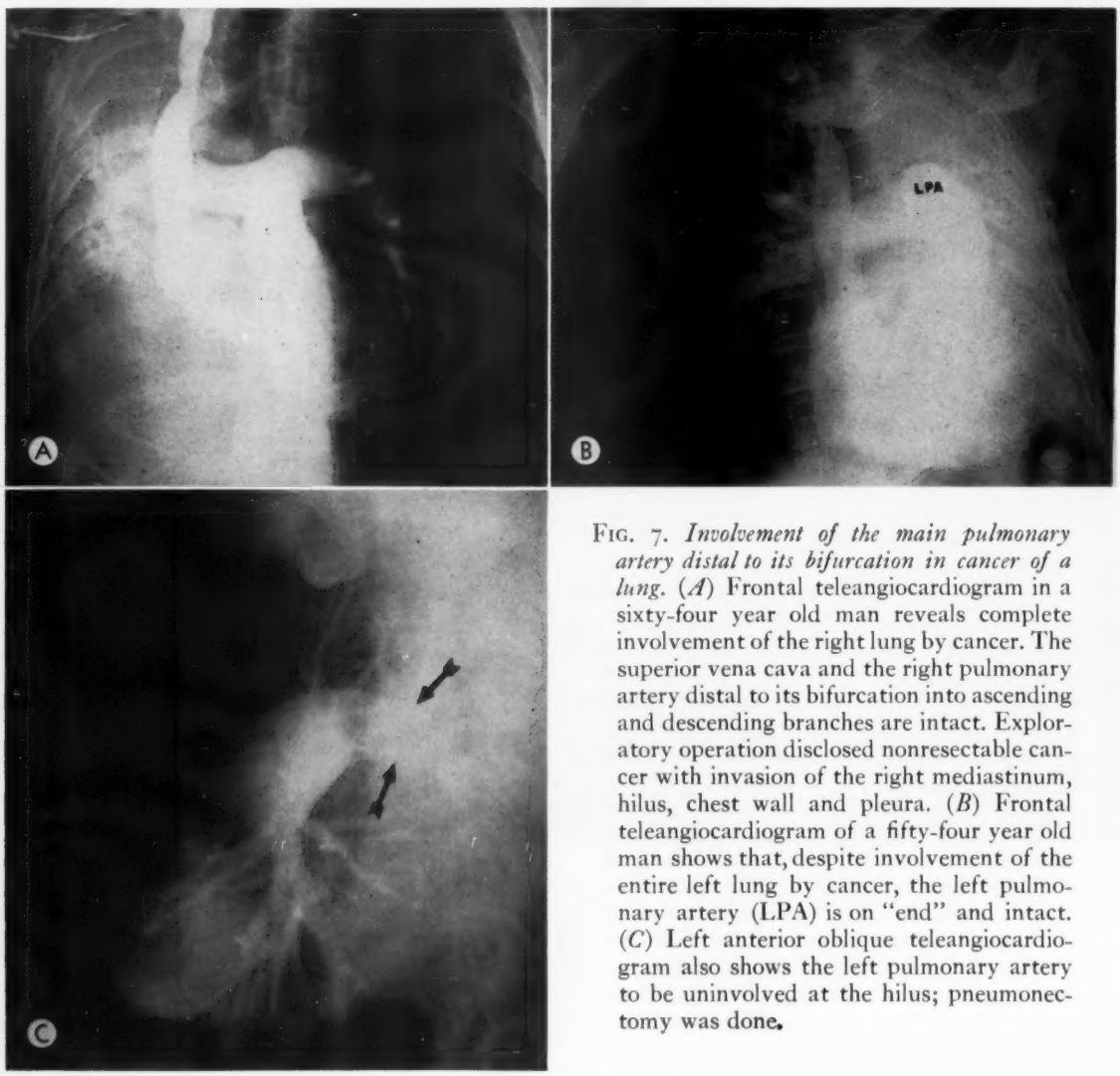

FIG. 7. *Involvement of the main pulmonary artery distal to its bifurcation in cancer of a lung.* (*A*) Frontal teleangiocardiogram in a sixty-four year old man reveals complete involvement of the right lung by cancer. The superior vena cava and the right pulmonary artery distal to its bifurcation into ascending and descending branches are intact. Exploratory operation disclosed nonresectable cancer with invasion of the right mediastinum, hilus, chest wall and pleura. (*B*) Frontal teleangiocardiogram of a fifty-four year old man shows that, despite involvement of the entire left lung by cancer, the left pulmonary artery (LPA) is on "end" and intact. (*C*) Left anterior oblique teleangiocardiogram also shows the left pulmonary artery to be uninvolved at the hilus; pneumonectomy was done.

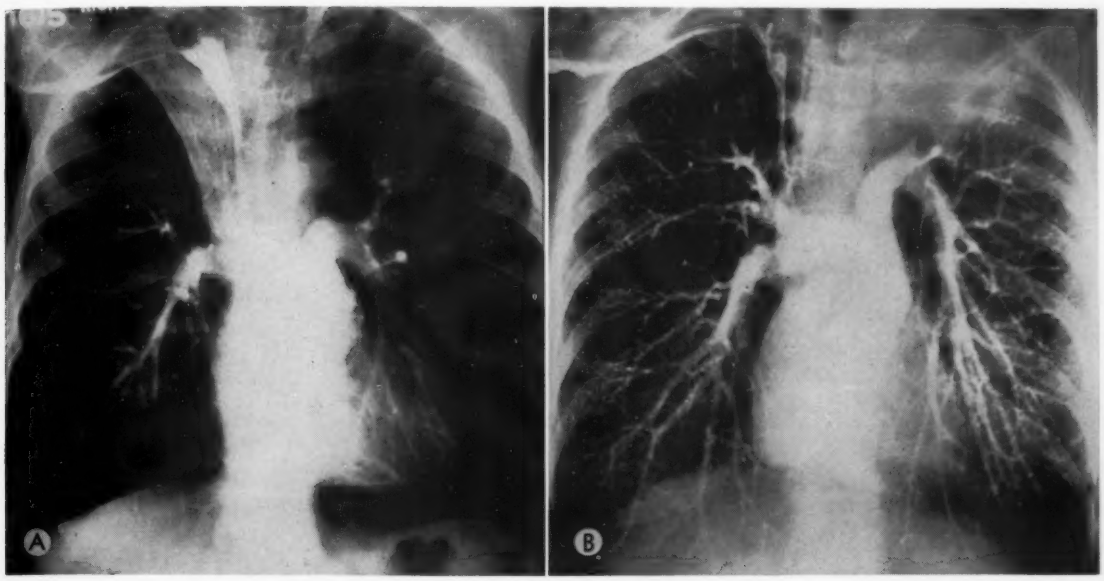

Fig. 8. *Segmental pulmonary arterial involvement in lung cancer.* (*A*) Frontal teleangiocardiogram of a sixty year old man shows atelectasis of the right upper lobe due to cancer at the origin of the bronchus. Operation was refused and improvement with resolution of atelectasis followed radiation therapy. There was a six year survival. (*B*) Frontal teleangiocardiogram of a sixty-two year old woman reveals complete occlusion of the left upper lobe pulmonary artery by a lung cancer. Exploratory operation confirmed the angiocardiographic findings but the tumor was too adherent to the chest wall to permit resection.

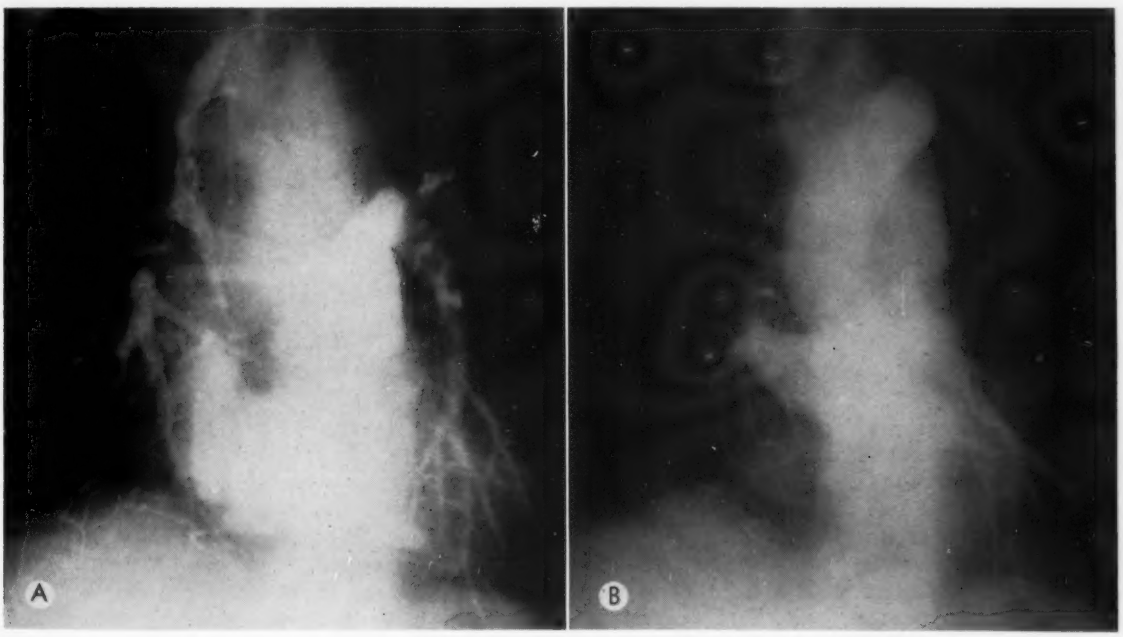

Fig. 9. *Pericardial, pulmonary arterial, left atrial and pulmonary venous involvement in lung cancer.* (*A*) Frontal serial angiocardiogram reveals superior vena caval stenosis and right pericardial invasion in a seventy year old man due to right middle lobe and hilar cancer. (*B*) Involvement of the right superior pulmonary vein and left atrium in the same patient. Because of angiocardiographic signs of inoperability, treatment was by roentgen rays; death occurred within a month.

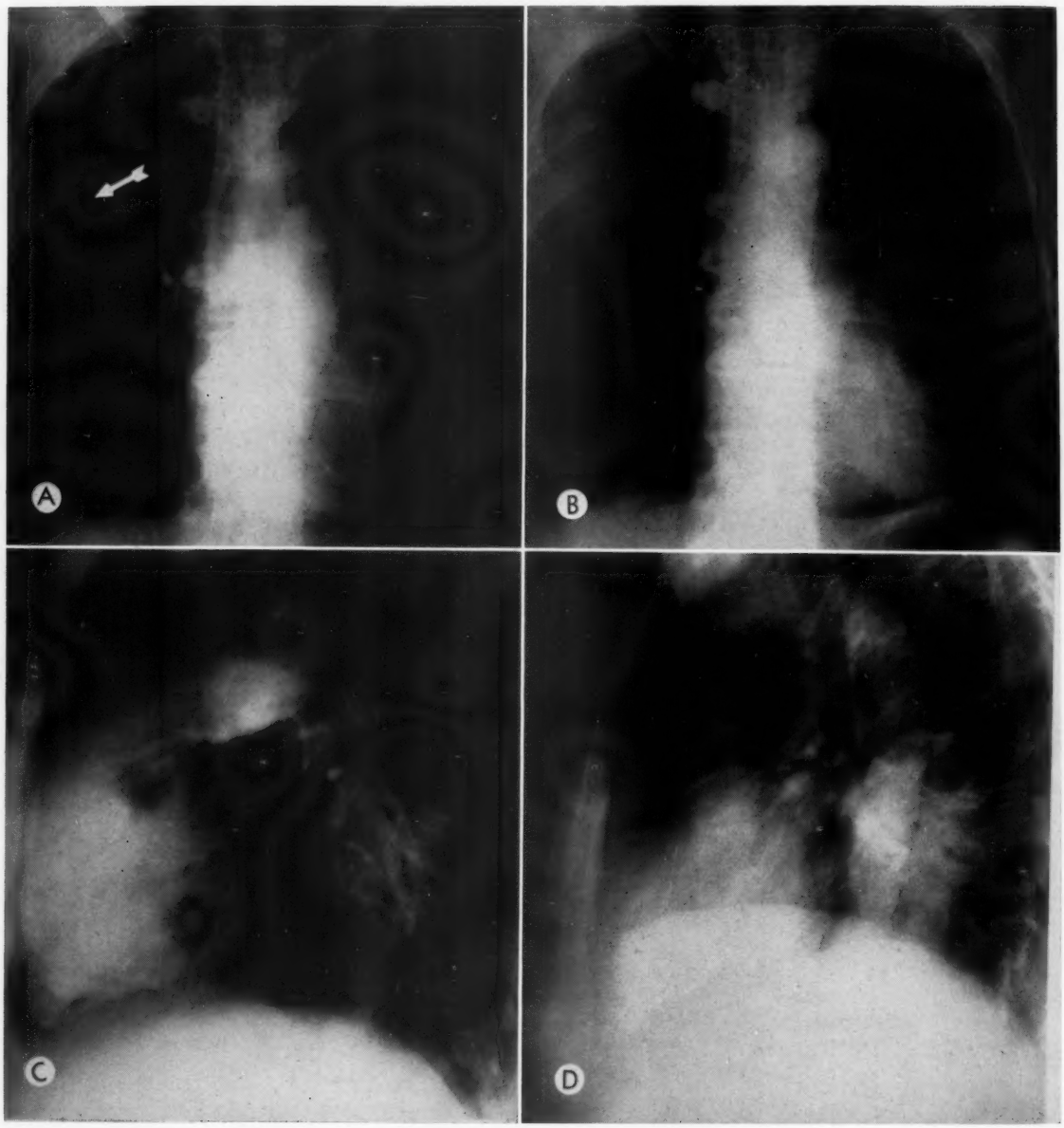

FIG. 10. *Angiocardiographic demonstration of "tumor vascular staining" in lung cancer.* (*A*) Frontal serial angiocardiogram at three seconds after injection of a sixty-six year old man with a peripheral cancer of the right upper lobe (arrow). The pulmonary arterial circulation appears uninvolved. (*B*) Later, at eight seconds after the left ventricle, aorta and brachiocephalic arteries were opacified, there was an increase of opacification of the tumor mass; pneumonectomy was performed but death occurred after three weeks. At autopsy, extensive emphysema of the left lung and cor pulmonale were found. (*C*) Lateral serial angiocardiogram at three seconds of a forty-seven year old woman with a left lower lobe cancer showing an intact pulmonary arterial blood supply. (*D*) At eight seconds, the tumor mass appeared opacified due to increased bronchial arterial circulation. Operation was not done because of metastases to the scalene lymph nodes.

when the lung cancer is confined to a segment or lobe, lobectomy in these patients might be tolerated.

SUMMARY AND CONCLUSIONS

Angiocardiographic study of 250 consecutive cases of primary cancer of the lung disclosed that 30 per cent involved the great vessels (superior vena caval and central pulmonary arterial) and were therefore nonresectable. These cases were chiefly due to hilar and mediastinal involvement.

In over 50 per cent of the cases, lobar and segmental pulmonary arterial involvement was demonstrated, thus permitting preoperative evaluation of the cardiovascular system. In some instances, because the vascular deformity was confined to a

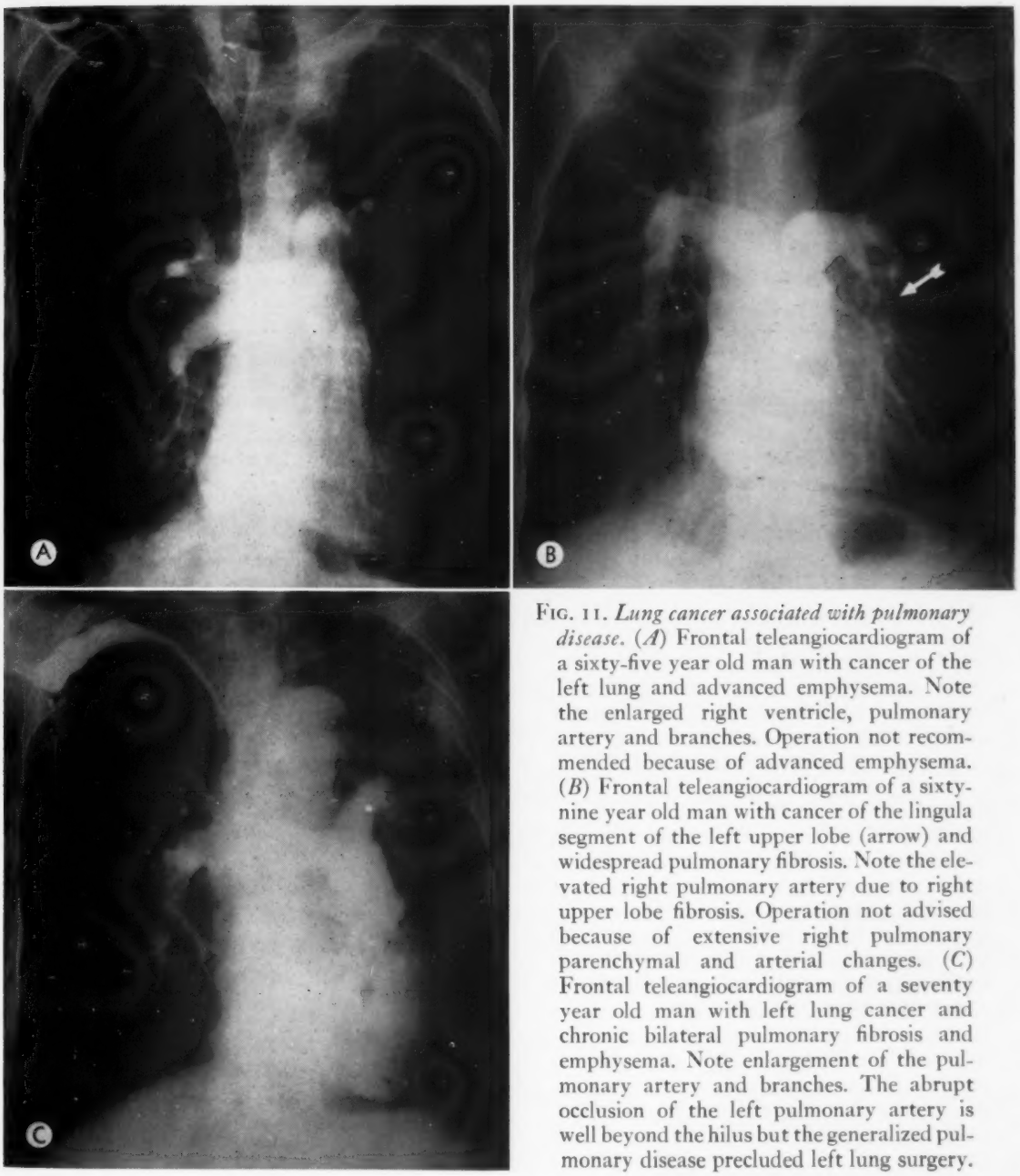

FIG. 11. *Lung cancer associated with pulmonary disease.* (*A*) Frontal teleangiocardiogram of a sixty-five year old man with cancer of the left lung and advanced emphysema. Note the enlarged right ventricle, pulmonary artery and branches. Operation not recommended because of advanced emphysema. (*B*) Frontal teleangiocardiogram of a sixty-nine year old man with cancer of the lingula segment of the left upper lobe (arrow) and widespread pulmonary fibrosis. Note the elevated right pulmonary artery due to right upper lobe fibrosis. Operation not advised because of extensive right pulmonary parenchymal and arterial changes. (*C*) Frontal teleangiocardiogram of a seventy year old man with left lung cancer and chronic bilateral pulmonary fibrosis and emphysema. Note enlargement of the pulmonary artery and branches. The abrupt occlusion of the left pulmonary artery is well beyond the hilus but the generalized pulmonary disease precluded left lung surgery.

segment or lobe, lobectomy rather than pneumonectomy seemed feasible. Finally, with bilateral pulmonary disease, angiocardiography provided a precise evaluation of the pulmonary circulation. Thus, pneumonectomy in a potentially respiratory crippled patient was avoided.

For the proper evaluation of the cardiovascular system in patients with lung cancer, scrupulous attention to the roentgenographic technique of angiocardiography is essential. Frontal (erect) angiocardiograms for visualization of the superior vena caval and pulmonary arterial circulation are mandatory. If there is involvement of the main branches of the pulmonary artery, oblique studies are also indicated. The right anterior oblique position should be used when there is deformity of the right pulmonary artery; the left view when the left pulmonary artery is involved. Then only will angiocardiography achieve its maximum usefulness in determining resectability and prognosis.

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ADDENDUM

Since this paper was submitted for publication, Lyons, H. A., and Vertova, F. (Angiocardiography: an aid for the early diagnosis of bronchogenic carcinoma. *Am. J. Med. Sci.*, 1958, *236*, 147–155) have reviewed the literature on angiocardiography in lung disease, especially cancer. They advocate that irregularities and occlusions of the pulmonary arterial tree be regarded as early signs of pulmonary cancer. Since many pulmonary diseases, notably tuberculosis, distort the pulmonary vascular system, caution against attributing such changes to cancer is advised.

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Vol. 81, No. 5

ROENTGEN FINDINGS IN ANEURYSM OF THE DESCENDING THORACIC AORTA*

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WITH the recent advances in vascular and thoracic surgery,[3,4,17] the roentgenologic differentiation of aneurysms from other intrathoracic lesions takes on added significance. In the past, little was to be offered to the patient with aortic aneurysm. Now, with a practical surgical solution to many of these vascular problems, the accurate preoperative diagnosis of an aneurysm is of great importance to the thoracic surgeon in outlining his surgical approach. The roentgen method of study[11,14] offers the surgeon maximal information regarding the location and probable nature of the lesion to be encountered. The scope of this study is limited to aneurysms of the descending thoracic aorta.

ETIOLOGY

Aneurysms of the aorta may be classified as follows:[1,7,8,9] (1) syphilitic; (2) arteriosclerotic; (3) traumatic; (4) congenital; (5) due to traction, usually associated with tuberculosis, or patent ductus arteriosus; (6) mycotic; and (7) secondary to coarctation of the aorta (poststenotic dilatation). Statistically, syphilis and arteriosclerosis account for the majority of all aortic aneurysms. Aneurysms of the thoracic aorta may originate in the ascending aorta, the aortic arch, and the descending thoracic aorta.

According to Maniglia and Gregory,[12] in an analysis of 6,000 consecutive autopsies, 100 cases of aortic aneurysms were found. Of this total number, only 8 cases were located in the descending thoracic aorta. In their series, 5 were syphilitic and 3 were arteriosclerotic. Whiteman and Mihaly,[18] in their series of 54 cases of thoracic aortic aneurysms, found that 35 were luetic and 19 arteriosclerotic. Ten of their cases were located in the descending thoracic aorta and of these 8 were arteriosclerotic and 2 were syphilitic. Freedman, Higley, and Hauser,[6] in a series of 150 thoracic aortic aneurysms, found 29 in the descending thoracic aorta. Epstein and Friedman[5] reviewed the literature and of 5,636 cases of aortic aneurysm, 320, or 5.7 per cent, were found in the descending thoracic aorta. The etiology was mentioned in 473 cases, and was syphilitic in 94.3 per cent. Under the age of forty-five, nonsyphilitic aneurysms are uncommon.[15]

REPORT OF CASES

CASE I. L.A., a fifty-three year old Caucasian male, was admitted on March 15, 1942 with chief complaints of pain in the left chest and weakness of the left arm of seven hours' duration. Since 1937, the patient had been hospitalized on three occasions for luetic aortitis and aneurysm. His blood pressure was 165/100. Serology was positive. The patient became comatose and expired shortly after admission. Autopsy findings included aneurysmal dilatation of the aorta 9 cm. from its origin, measuring 5.5 cm. in diameter. This aneurysm caused erosion of the sternum. A second aneurysm, measuring 9×11 cm., was found in the descending thoracic aorta. This aneurysm caused erosion of the adjacent vertebrae. Chest roentgenograms (Fig. 1, *A* and *B*) in the posteroanterior and left anterior oblique views revealed a large saccular aneurysm of the descending thoracic aorta with erosion of vertebrae. The aneurysm of the ascending aorta was also seen.

CASE II. D.O., a forty year old Negro male, was admitted on January 18, 1938 with a chief complaint of pain in the right flank of one month's duration. The patient was treated for syphilis and pulmonary tuberculosis from 1938 to 1940. The blood pressure was 140/110. Serology was positive. Sputum was positive for acid fast bacilli. A posteroanterior roentgenogram of

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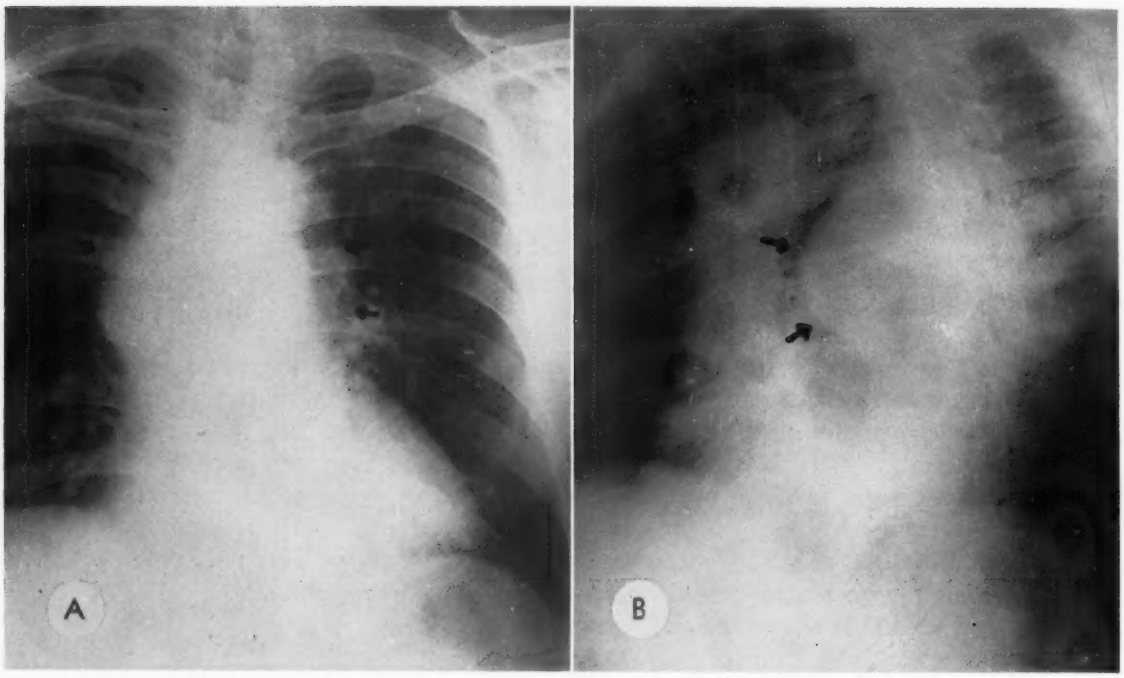

FIG. 1. Case I. (*A* and *B*) Roentgenograms showing the saccular aneurysm of the descending thoracic aorta with vertebral erosion. Second aneurysm of the ascending thoracic aorta is also visualized.

FIG. 2. Case II. Roentgenogram showing the saccular aneurysm of the descending thoracic aorta in the right cardiophrenic angle. Note also the advanced pulmonary tuberculosis.

the chest (Fig. 2) revealed extensive bilateral cavitation. Suspected aneurysm of the descending thoracic aorta presented in the right cardiophrenic angle. The patient expired on October 4, 1940. Autopsy revealed a large saccular aneurysm of the descending thoracic aorta just above the diaphragm measuring 14 cm. in diameter, with erosion of adjacent vertebrae.

CASE III. D.H.M., a fifty-five year old Negro male, was admitted on August 1, 1950 for treatment of a peptic ulcer. He was also found to have a goiter. Gastric surgery was performed and in December, 1951 he was re-admitted for a thyroidectomy. Chest fluoroscopy to rule out substernal thyroid revealed a triangular density in the left hilus, thought to be vascular in origin. A posteroanterior roentgenogram of the chest (Fig. 3*A*) revealed a rounded soft tissue mass extending from the left hilus. The trachea was deviated to the right by a substernal thyroid. The blood pressure was 140/100. Serology was negative. A subtotal thyroidectomy was performed in 1952 for a nontoxic thyroid adenoma. A retrograde aortogram (Fig 3*B*) on July 9, 1952 revealed no evidence of passage of contrast medium from the aorta into the adjacent

mass. Bronchoscopy was negative. A thoracotomy on August 19, 1952 disclosed minimal diffuse dilatation of the entire thoracic aorta. In the region of the sixth thoracic vertebra, extending from the posterolateral aspect of the aorta, there was a local aneurysmal dilatation of the descending thoracic aorta measuring approximately 3×4 cm. The aneurysm was considered to be congenital. No surgical treatment was undertaken. Final admission on January 2, 1953 showed hypertensive heart disease and cerebral thrombosis. He expired on January 17, 1953.

Case iv. D.R., a forty-seven year old Mexican male, was admitted on April 18, 1953 with a chief complaint of left corneal ulceration. Routine photofluorography revealed a mass in the left hilus. Posteroanterior and left lateral roentgenograms (Fig. 4, *A* and *B*) revealed a mass interpreted as a round circumscribed neoplasm located in the apical portion of the left lower lobe. A bronchogenic tumor or neurogenic neoplasm was considered. The blood pressure was 180/98. Serology was negative. Bronchoscopy revealed no tumor. Thoracotomy on October 16, 1952 disclosed a pulsating aneurysm, 4 cm. in diameter, in the mid-descending thoracic aorta. Twenty feet of stainless steel wire was introduced into the aneurysm and then wrapped in cellophane.

Case v. J.W.F., a thirty-one year old Caucasian male, was admitted on October 21, 1955. His chief complaint was of recent central chest pain radiating to the right shoulder. The pain was sharp and agonizing. He had had a similar attack three to four months previously. The blood pressure was 142/74. There were no positive physical findings. No serology was recorded. A routine posteroanterior roentgenogram of the chest was interpreted as demonstrating a possible bronchogenic cyst or a vascular anomaly. An anteroposterior laminagram and a left anterior oblique roentgenogram of the chest (Fig. 5, *A* and *B*) revealed curvilinear calcification in the wall of the aneurysm. Thoracotomy on November 17, 1955 showed aneurysmal dilatation of the anterior aspect of the descending thoracic aorta, just distal to the

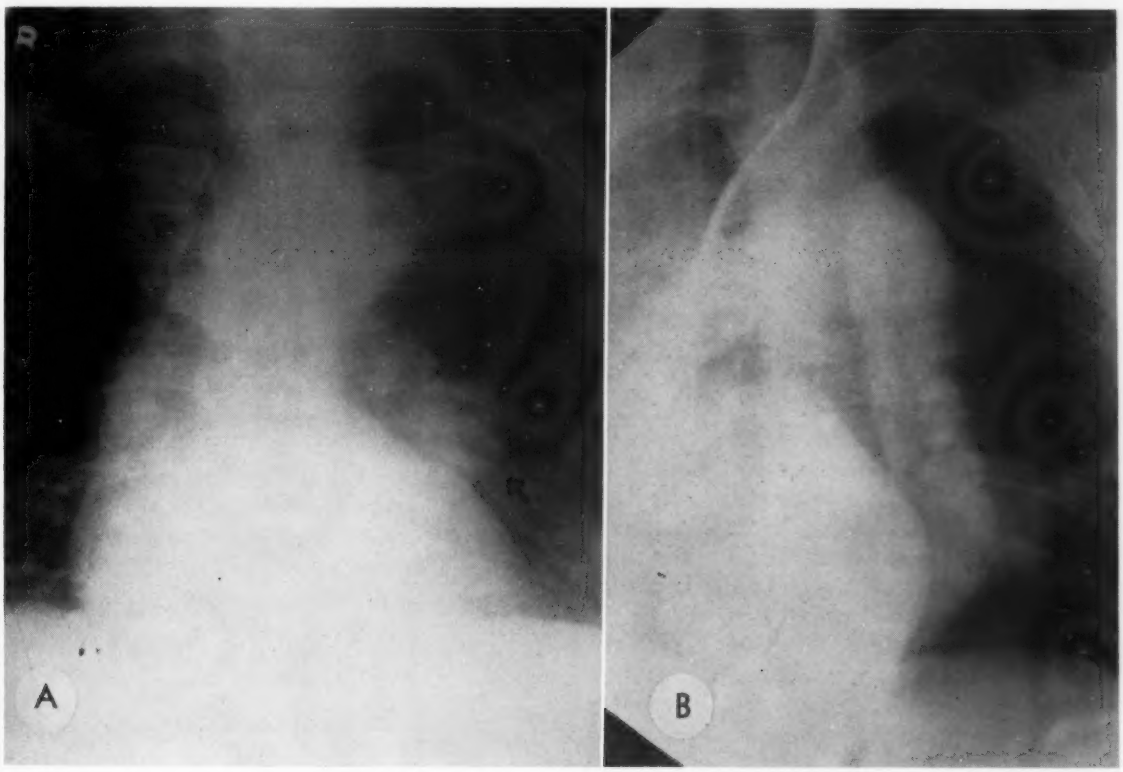

Fig. 3. Case iii. (*A*) Roentgenogram showing the saccular aneurysm of the descending thoracic aorta in the left infrahilar area. (*B*) Retrograde aortogram reveals nonfilling of aneurysm due to laminated clot.

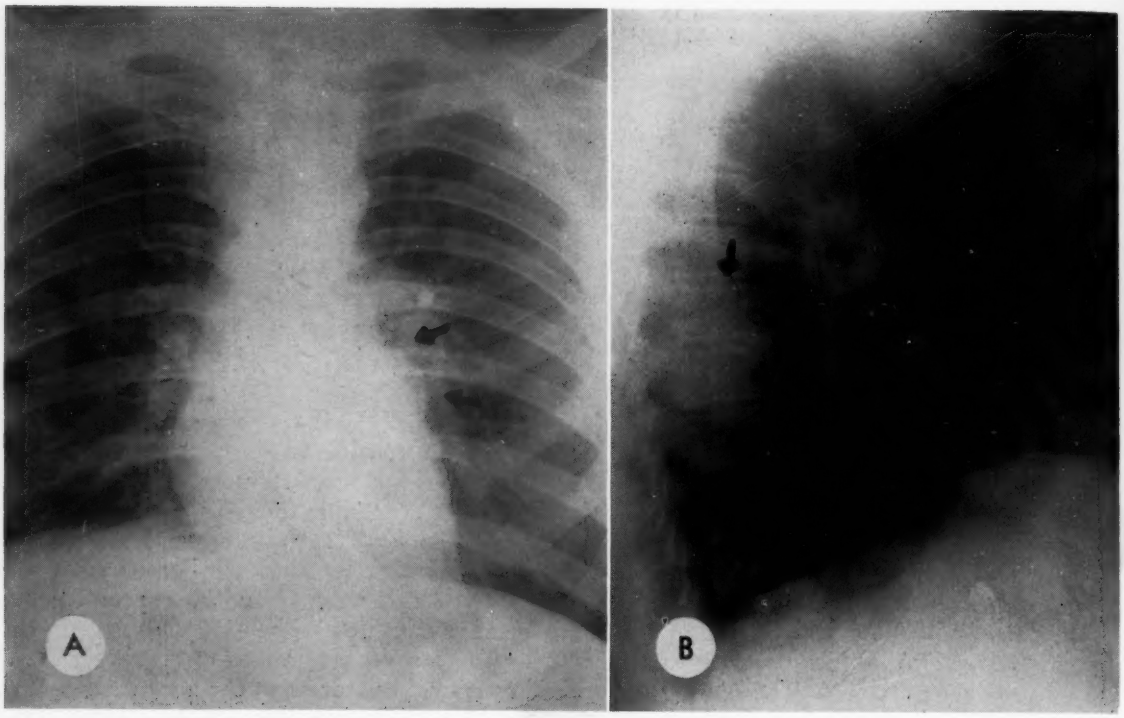

FIG. 4. Case IV. (*A*) Roentgenogram showing the saccular aneurysm of the descending thoracic aorta in the left infrahilar area. The aneurysm mimics a parenchymal tumor in the apical segment of the left lower lobe on lateral view (*B*).

arch, measuring 5 cm. in greatest diameter. This was thought to be congenital in origin. No surgical correction was attempted. He was last seen on January 18, 1956 and was asymptomatic.

CASE VI. W.E.C., a forty-five year old Negro male, was admitted on March 23, 1949 with chief complaints of stabbing low back pain of two months' duration and weakness from the waist down. The blood pressure was 140/90. Serology was positive. The clinical impression was that of a possible spinal cord tumor or tabes dorsalis. Chest fluoroscopy demonstrated a large semicircular mid-thoracic soft tissue mass in the region of the descending thoracic aorta. There was no evidence of pulsation. Erosion of T8 to T11 was noted. The impression from fluoroscopy and subsequent chest roentgenograms (Fig. 6, *A* and *B*) was that of aortic aneurysm versus neurofibroma. Thoracotomy on May 3, 1949 revealed a saccular aneurysm, measuring 10×8 cm., on the posterior surface of the descending thoracic aorta. This was wrapped with polyethylene tubing and a mixture of talc and glue. The patient, last seen on August 9, 1949, was symptomatically improved.

CASE VII. T.D., a seventy year old Caucasian male, was admitted on February 15, 1950. He was semicomatose and no history was obtainable. The past history revealed a diagnosis of tabes dorsalis in 1932 with numerous subsequent tabetic crises. Numerous blood and spinal serologies were negative. The blood pressure was 196/130. The patient had a positive left Babinski reflex. Chest roentgenograms (Fig. 7, *A* and *B*) showed extensive calcification of the entire thoracic aorta and a dilatation of the mid-descending thoracic aorta, measuring 6×9 cm., with a curvilinear calcification in its wall. He expired on February 18, 1950. Autopsy disclosed a fusiform dilatation of the ascending aorta and a saccular dilatation of the descending thoracic aorta, measuring 5 cm. in its greatest diameter, containing antemortem thrombus.

SYMPTOMS AND PHYSICAL FINDINGS

Substantial reliance cannot be placed on classic symptoms or physical findings in cases of aortic aneurysms. Many are asymptomatic and are discovered incidentally on routine chest roentgenograms.

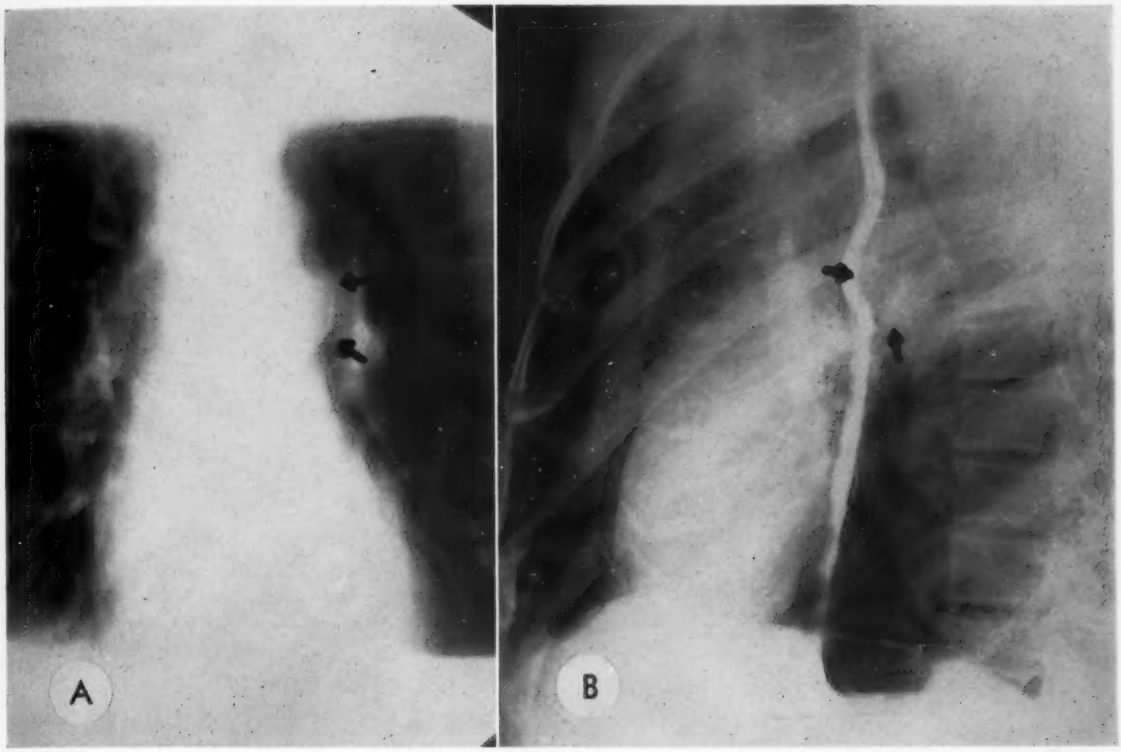

FIG. 5. Case V. (*A*) Laminagram showing curvilinear calcification in the saccular aneurysm of the upper descending thoracic aorta. (*B*) Left anterior oblique roentgenogram showing the aneurysm and the deviation of the barium-filled esophagus.

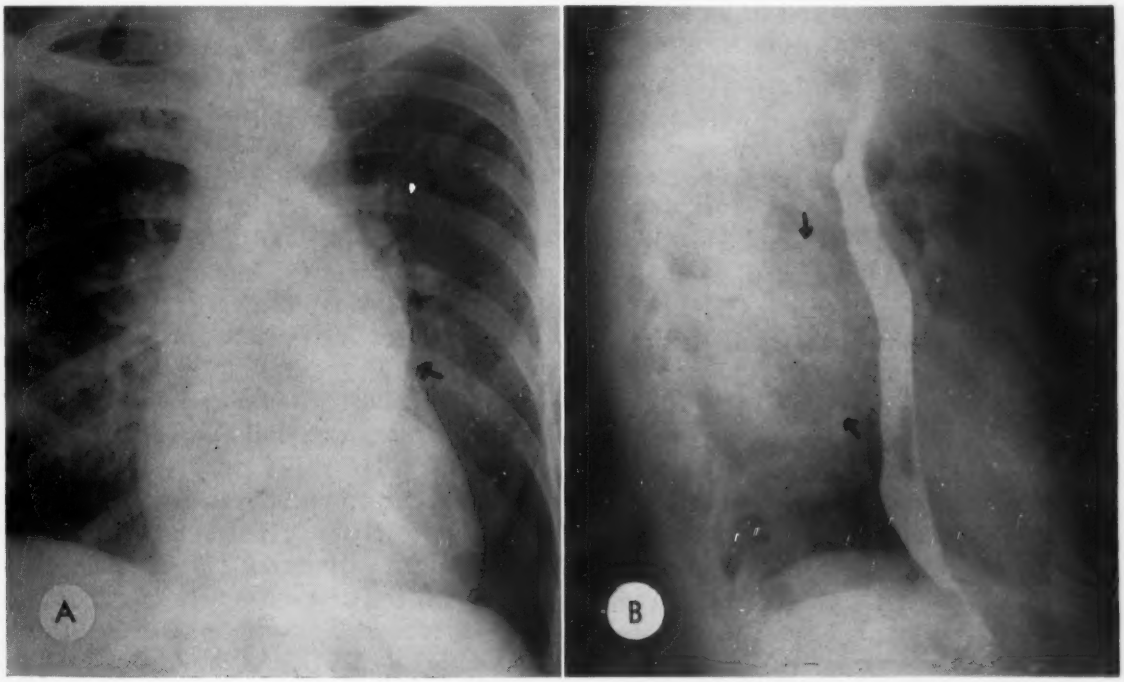

FIG. 6. Case VI. (*A* and *B*) Roentgenograms showing the large saccular aneurysm of the descending thoracic aorta eroding vertebrae T8–11.

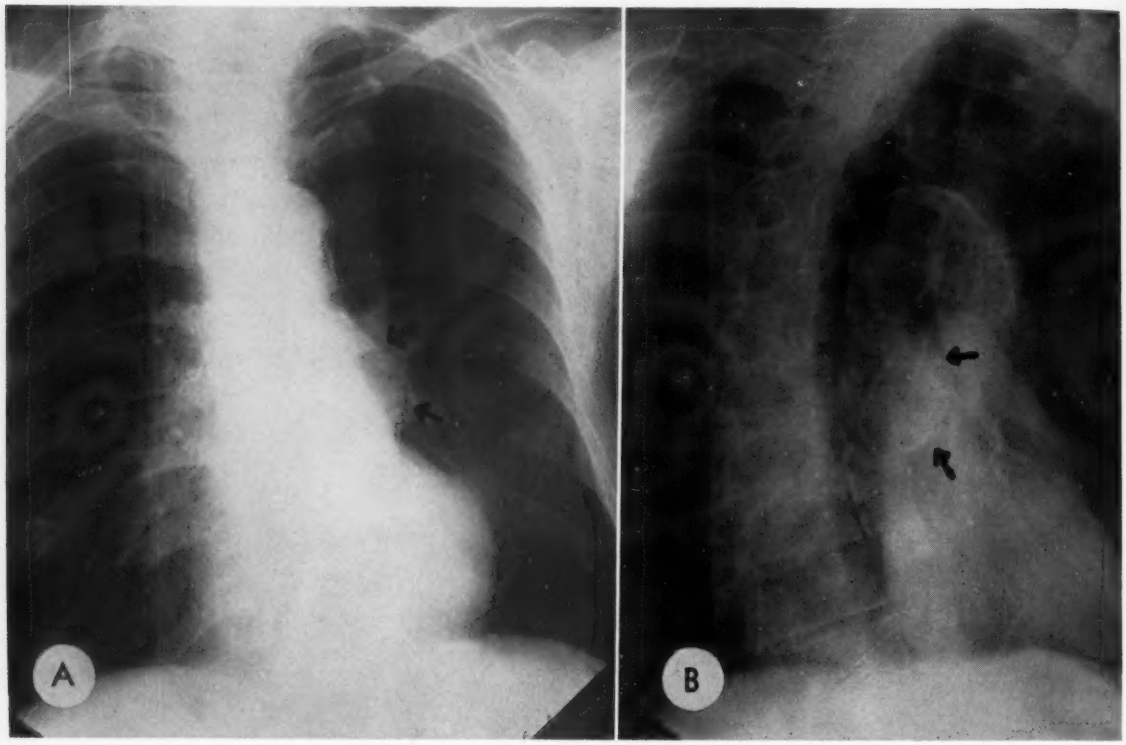

FIG. 7. Case VII. (*A* and *B*) Roentgenograms showing the saccular aneurysm of the descending thoracic aorta with curvilinear calcification in its wall. Note also the extensive calcification of the entire thoracic aorta.

ROENTGEN FINDINGS AND DIFFERENTIAL DIAGNOSIS

On the posteroanterior roentgenogram of the chest, aneurysms of the descending thoracic aorta may present in the left or right hilar areas or in the right cardiophrenic angle.

Aneurysms which present from the left (Cases III, IV, V, VI and VII) or right (Case I) hilar areas must be differentiated mainly from the following: (1) mediastinal and hilar lymphadenopathy from any cause; (2) bronchogenic carcinoma;[13] (3) mediastinal tumors[2,16] and (4) pulmonary artery dilatation.

Those aneurysms presenting in the right cardiophrenic angle (Case II) require differentiation from the following: (1) tortuous descending thoracic aorta; (2) hiatal hernia; (3) pericardial cyst; (4) pleural or diaphragmatic tumors; (5) achalasia and (6) hernia of the foramen of Morgagni.

The lateral and oblique chest roentgenograms will in most instances reveal that the mass, if due to aneurysm of the descending thoracic aorta, is posterior. Since we are limiting our discussion to aneurysms of the descending thoracic aorta, the differential diagnosis is primarily one of posterior and middle mediastinal masses. Therefore, in the above list of possibilities, one must also include: (1) neurogenic tumors; (2) paraspinal abscess (Pott's disease); (3) meningocele; (4) enterogenous cysts; (5) cardiac aneurysms; (6) posterior mediastinal or pleural fluid collections; (7) granulomatous or neoplastic involvement of posterior lung parenchyma or thoracic wall and (8) esophageal tumors.

The roentgen findings in aneurysm of the descending thoracic aorta may be briefly summarized as follows: (1) the mass must stay with the descending thoracic aorta in all projections;[10] (2) the mass may or may not have calcification within its walls (Case V, Fig. 5*A* and Case VII, Fig. 7, *A* and *B*); (3) the esophagus may be displaced if the aneurysm reaches sufficient proportions (Case V, Fig. 5*B* and Case VI, Fig. 6*B*); (4)

there may be erosion of adjacent vertebrae or ribs (Case I, Fig. 1*B* and Case VI, Fig. 6*B*); (5) at fluoroscopy, the presence or absence of expansile pulsations (Case VI) may be difficult to evaluate,[11] and differentiation from transmitted pulsations may be impossible; (6) laminagraphy (Case V, Fig. 5*A*) may aid to visualize calcification in the wall of the aneurysm and/or erosion of vertebrae or ribs and (7) angiocardiography[2,16] or retrograde aortography (Case III, Fig. 3*B*) may further aid to outline an aneurysm. However, it must be pointed out that saccular aneurysms frequently do not fill. This may be due either to the narrow opening of the aneurysm or to the presence of a laminated clot within the aneurysmal sac.

SUMMARY

Seven cases of aneurysm of the descending thoracic aorta are reported. Three cases were syphilitic and 4 were arteriosclerotic.

Four cases were subjected to thoracotomy. Of these 4, 2 were correctly diagnosed preoperatively; the third was thought to have a bronchogenic cyst and the fourth a bronchogenic carcinoma.

In the 3 remaining cases, the correct diagnosis was made on routine roentgenographic examinations.

Five of the 7 cases exhibited on the posteroanterior roentgenogram of the chest a mass in the hilar area, one on the right side and the other 4 on the left side. In another case the aneurysm simulated an enlarged pulmonary artery segment in the posteroanterior chest roentgenogram. The last case showed a mass in the right cardiophrenic angle.

Roentgen studies, including retrograde aortography, angiocardiography and laminagraphy, will, in most instances, establish the diagnosis of aneurysm of the descending thoracic aorta.

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ROENTGEN SIGNS OF CONTRAST MATERIAL DISSECTION OF AORTIC WALL IN DIRECT AORTOGRAPHY*

By DELBERT E. BOBLITT, M.D., MELVIN M. FIGLEY, M.D., *and* EARL F. WOLFMAN, JR., M.D.
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TRANSLUMBAR aortography has become a widely used procedure of considerable value in the clinical appraisal of occlusive vascular disease, hypertension of renal vascular disease origin, intra-abdominal masses of obscure nature and location, and abdominal and pelvic arteriovenous malformations. However, it is not a simple diagnostic procedure to be chosen without careful consideration of the information to be provided by it that is unobtainable by other means.

To other reports concerning complications of translumbar aortography,[1–4,8,9] we add our experiences with the inadvertent injection of all or a portion of the contrast material into the wall of the aorta rather than into the lumen of the vessel itself. During such injections, the bevel of the needle presumably lies partly within the aortic wall, or the contrast material has access to the aortic wall through the hole produced by the needle puncture. Since many persons upon whom aortography is performed have serious vascular disease, it is easy to separate the diseased medial layer of the artery with either the needle tip or the jet stream of injected contrast material.

Large amounts of contrast material intramurally deposited may cause rapid occlusion of major aortic branches and give rise to symptoms immediately; or a false extraluminal channel may be formed which allows an inflow of blood from the main aortic lumen to occur. The clinical manifestations of such a dissection can appear at variable periods of time after the intramural injection and are related in all probability to the time interval required for blood to enter the falsely produced channel.

MATERIAL AND INCIDENCE OF COMPLICATIONS

A series of 425 translumbar aortographies in 390 patients performed at the University of Michigan Hospital has been reviewed. Examinations done by retrograde catheter injection technique have not been included. The procedures have been done with a single short bevel No. 16 or No. 18 needle under either spinal or intravenous pentothal anesthesia with rapid manual injection of 70 per cent urokon (sodium acetrizoate) as contrast medium. For the most part manual changing of cassettes has been employed. A test dose of 5 cc. of contrast medium has been employed routinely to ascertain needle position, and no injections were made unless a pulsatile flow of blood from the needle hub was seen prior to injection. The average total dose of contrast medium has been between 20 and 30 cc., though on occasion the total dosage has been extended through more than one injection to 60 cc. In one instance a total of 100 cc. was injected. The dosage used has been reduced over the past year so that a total dose of over 25 cc. now seems unnecessary. Frequently, less will suffice.

In this series intramural injections of varying amounts have occurred in 42 patients, an incidence of approximately 10 per cent. Some of the intramural injections so designated consisted of only 1 cc. or less, while some amounted to a large portion of the injected volume. The only serious or significant complications in the entire series were in patients having intramural injec-

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tions. There were 3 deaths, 2 of them with proved dissecting aneurysms surrounding the renal arteries and producing uremia. These aneurysms were observed between the middle and outer thirds of the media, the same location in which spontaneous dissecting aneurysms commonly occur. The third case, previously reported from this hospital,[2] appears in retrospect to have had a short strategically located intramural injection at the level of the renal arteries. No autopsy was permitted in this case so that one can do no more than suspect that the intramural injection may have been a significant factor in the patient's uremia and death. In this case, receiving the largest amount of contrast material ever used (100 cc.), it is difficult to avoid attributing death to the excessive volume of contrast material. However, only 40 cc. of that amount perfused the kidneys in high concentration, the remaining amount being injected at a puncture site low in the aorta. In this respect it is pertinent to note that Kincaid and Davis,[7] doing a large volume of aortographic work, frequently use large amounts of contrast material without apparent complications. Therefore, were the needle in its proper position in this case, the amount of contrast material may not have been as important a factor as one would immediately suspect.

Details of the clinical aspects of the 3 fatalities are covered in a separate publication.[12]

ROENTGEN SIGNS OF INTRAMURAL CONTRAST MATERIAL

The cardinal sign of an intramural injection is a sharply demarcated dense collection of contrast medium showing no evidence of mixing with nonopacified blood. The column of contrast material equals or only slightly exceeds the diameter of the vessel injected. There may be no filling of aortic branches if the injection is entirely intramural as shown in Figure 1*A*, or a good quality aortogram may be obtained if only a small portion has been deposited in the aortic wall, as demonstrated in Figure 4*A*. Should part of the contrast medium dissect back into the aortic lumen or rupture extramurally (Fig. 1*B*), a contrast material cast of the aortic wall may persist. If part of the injection is intraluminal, the same phenomenon may be observed as the intraluminal portion is swept downstream (Fig. 3*C* and 4*B*).

In uncomplicated aortography, the intraluminal contrast material is rapidly dispersed by the swift-moving bloodstream and extends proximally in the aorta only a few centimeters above the needle puncture site. Contrast medium more than the usual distance above the needle should arouse one's suspicion concerning an intramural injection. If the column is, in addition, sharply demarcated and abnormally dense, one has positive evidence of its intramural location. On occasion, this appearance is observed in a patient who has good palpable femoral pulses. In such a patient with a large deposit of contrast material seemingly in the aorta proximal to the needle, failure to observe contrast medium in the distal aorta and its terminal branches is further definite evidence that something has gone awry with the injection. This is especially true if no extensive collateral circulation is seen (Fig. 2*A*).

Additional evidence of an intramural injection is often noted on the delayed roentgenograms. Here one sees the effects of occlusion of aortic branches (either by the intramural injection or the resultant dissecting aneurysm) if contrast medium enters the affected branch before occlusion occurs. The viscus supplied by the occluded branch is persistently opacified by contrast material trapped within it as a result of the loss of normal perfusion pressure. This phenomenon is illustrated in Figure 2*B* by the persistent nephrogram obtained three hours following an aortogram that produced a dissecting aneurysm, occluding both renal arteries. The patient died of uremia in four days. Figure 2C shows the autopsy evidence of the dissecting aneurysm.

Figure 3*D* demonstrates the opacified

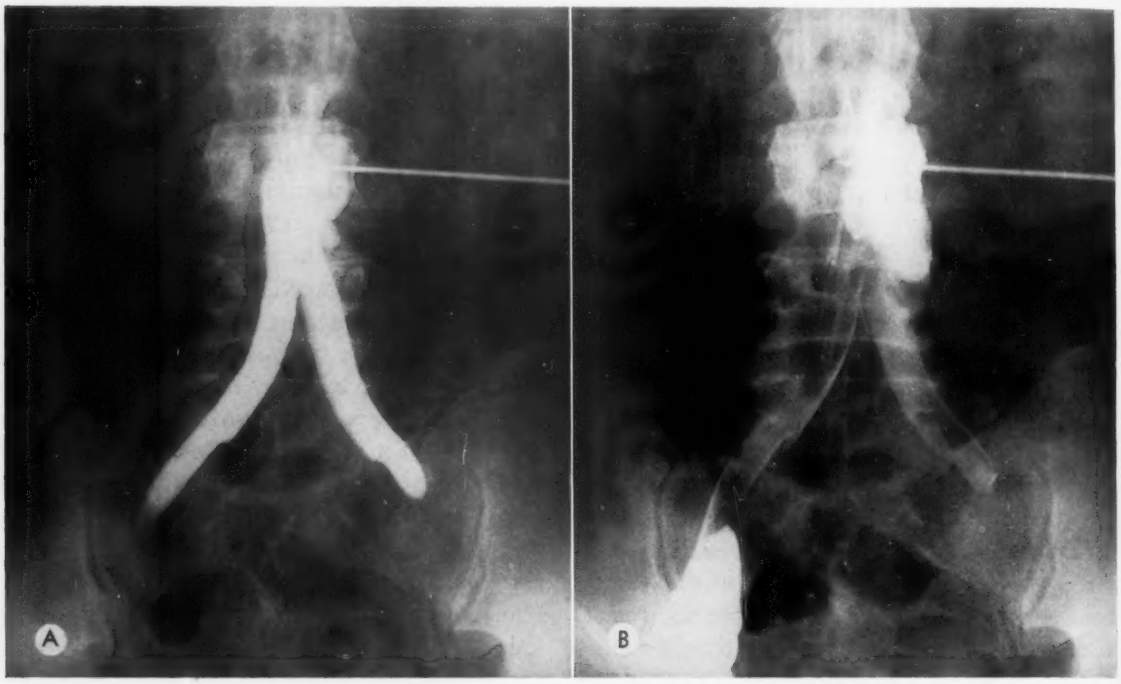

Fig. 1. (*A*) Intramural injection into the outer third of the media of the aorta. Note absence of filling of the aortic branches and some extramural extravasation at the puncture site. The total volume of injection was 15 cc. (This case is illustrated through courtesy of VA Hospital, Ann Arbor, Mich., and is not part of the statistical series.) (*B*) Aortogram taken six to eight seconds after *A*. The contrast material has dissected back along its original path and has ruptured externally around the right external iliac artery. This forty-six year old man died of ventricular fibrillation ninety minutes following aortography and was found to have unusually severe coronary artery disease at autopsy. (*A.M.A. Arch. Surg.*—Wolfman and Boblitt, April, 1959.)

rectal wall, which is another example of trapped contrast material. In this patient, who also died of uremia from a dissecting aneurysm surrounding the renal arteries, the inferior mesenteric artery was occluded. However, terminal branches of the hemorrhoidal arteries had apparently allowed perfusion of the rectum immediately prior to the occlusion, and contrast material was accordingly trapped therein. The resulting stasis of contrast material in all likelihood abetted the ischemic insult inasmuch as the entire rectum was completely necrotic at postmortem examination.

Intramural injections can be easily differentiated from the irregularly dispersed completely extramural or periaortic injections. In our experience intramural injections are observed more frequently than periaortic injections, which is easily understandable when one considers that an injection is not made unless some sort of pulsatile flow from the needle is seen prior to injection, indicating that the bevel of the needle has at least partial access to the aortic lumen. Many of the intramural injections were accompanied by extramural extravasations of varying amounts.

On occasion, contrast material in the aortic lumen is sharply demarcated distally in the presence of a complete aortic block. But here the resemblance to an intramural injection ends because evidence of collateral circulation will be obvious and will indicate that the abruptly terminated column is intraluminal and proximal to a complete block rather than in the aortic wall.

DISCUSSION

Intramural injection of contrast medium during direct aortography, while undoubtedly recognized by other workers, has been

given little attention in the literature. In a comprehensive questionnaire survey by McAfee[9] of 13,207 abdominal aortographies from 301 institutions in the United States, intramural injection or extravasation of contrast medium was listed as a minor complication, ranging in incidence in the 96 institutions reporting it from 1 to 20 per cent. In over half of these, the incidence was between 5 and 10 per cent, which broadly agrees with our own figure. (It is not stated definitely whether all workers have classified intramural and extramural injections separately.) However, our opinion is at variance with the designation of it as a minor complication.

This subject has been discussed in a most worthwhile paper by Gaylis and Laws,[3] who noted a few uncomplicated intramural injections and were able to reproduce an

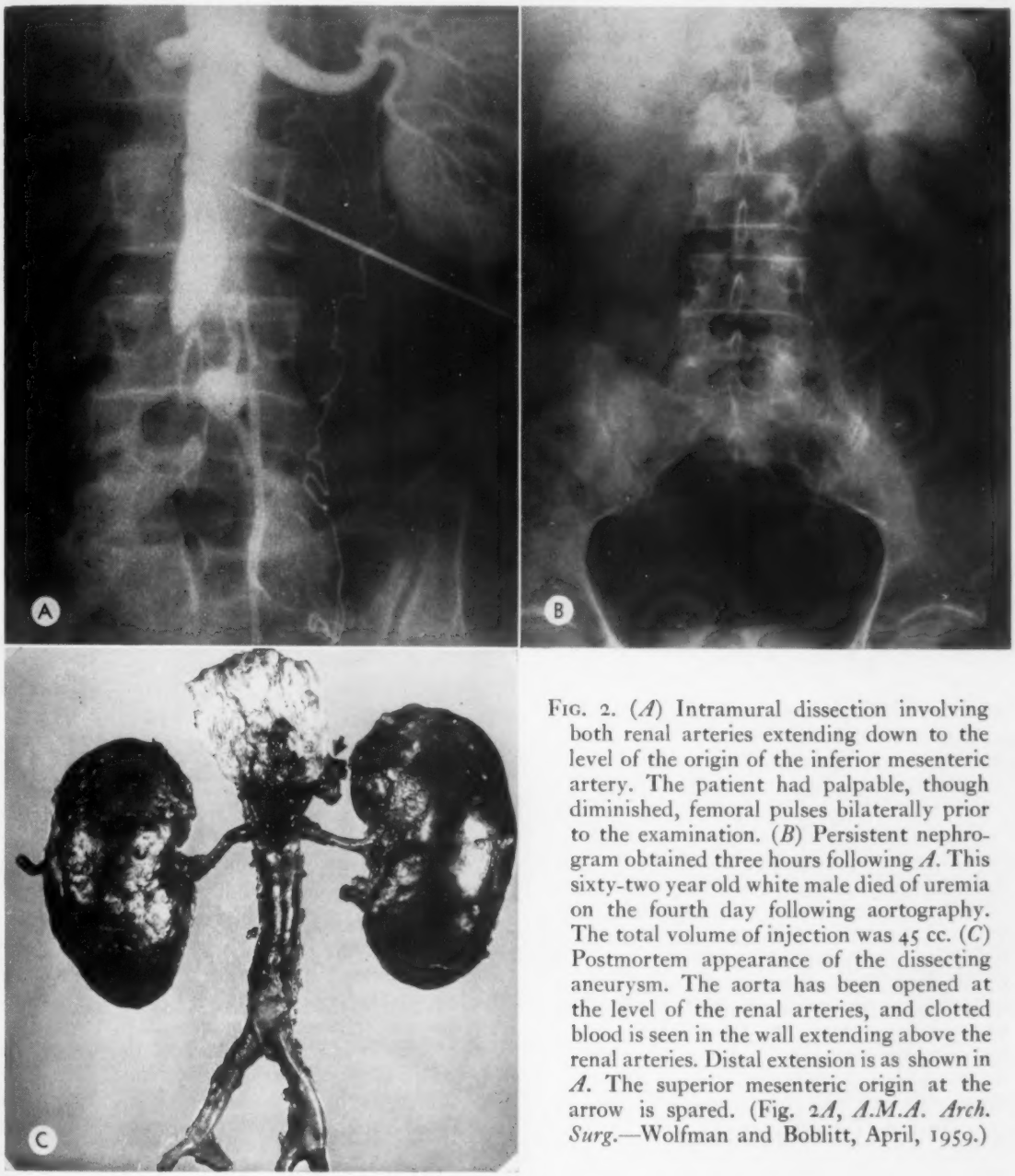

Fig. 2. (*A*) Intramural dissection involving both renal arteries extending down to the level of the origin of the inferior mesenteric artery. The patient had palpable, though diminished, femoral pulses bilaterally prior to the examination. (*B*) Persistent nephrogram obtained three hours following *A*. This sixty-two year old white male died of uremia on the fourth day following aortography. The total volume of injection was 45 cc. (*C*) Postmortem appearance of the dissecting aneurysm. The aorta has been opened at the level of the renal arteries, and clotted blood is seen in the wall extending above the renal arteries. Distal extension is as shown in *A*. The superior mesenteric origin at the arrow is spared. (Fig. 2*A*, *A.M.A. Arch. Surg.*—Wolfman and Boblitt, April, 1959.)

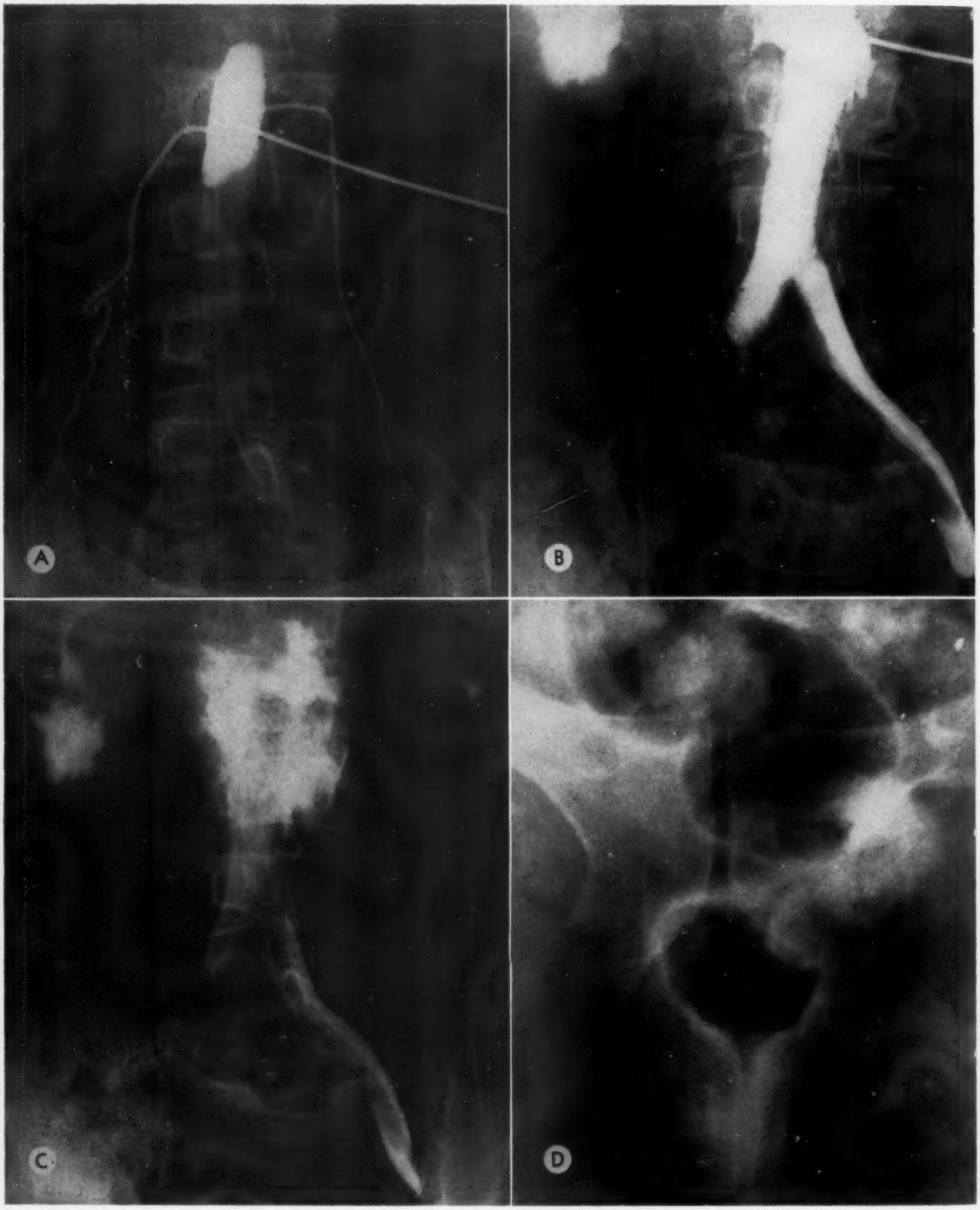

FIG. 3. (*A*) The intramural injection was interpreted erroneously as an aortic obstruction, and accordingly two additional injections were made. Final injection shown in *B*. The total volume injected was 45 cc. (*B*) In addition to the large intramural deposit of contrast material and the meager fill of the aortic branches, note the dense collection of the opaque medium in the lower pole of the right kidney. Perhaps this is distal to an atheromatous embolus occurring just after the contrast medium entered. A localized infarct resulted. (*C*) The dissection did not involve the renal arteries immediately because the patient did not show renal function impairment until the eighth day. This fifty-one year old man died of uremia on the twelfth day following aortography, both renal arteries being involved in a dissecting aneurysm. (*D*) Opacification of the entire rectal wall. The patient complained bitterly of rectal tenesmus as spinal anesthesia wore off, and rectal bleeding developed the following day. The rectum was necrotic at autopsy. (Fig. 3, *A* and *B*, *A.M.A. Arch. Surg.*—Wolfman and Boblitt, April, 1959.)

identical appearance in an aorta which was removed from another patient at autopsy, perfused in a Melrose heart-lung machine, and injected with contrast medium, the bevel of the needle being in contact with both the aortic lumen and the aortic wall. Our material confirms their very accurate supposition that on occasion an intramural injection could produce serious sequelae.

As these authors have pointed out, many of the common serious complications of direct aortography—renal, neurologic, and gastrointestinal—are identical to those associated with spontaneously occurring dissecting aneurysms of the aorta. Since we have observed that one can inadvertently produce a dissecting aneurysm during direct aortography, this is not surprising. Furthermore, just as spontaneous dissecting aneurysms do not always produce occlusions of major vascular trunks, neither do such occlusions invariably occur in dissecting aneurysms resulting from aortography. Examples of such uncomplicated intramural injections are reproduced in Figures 5, 6 and 7. Of our 42 intramural injections, only 3, or less than 10 per cent, were attended by significant ill effects.

It is an understandable tendency to attribute death of a viscus following angiography to contrast medium toxicity, especially if one observes perfusion of it through the involved organ in great concentration. The toxicity of the various iodinated compounds used in angiography has been demonstrated experimentally[6,10] as

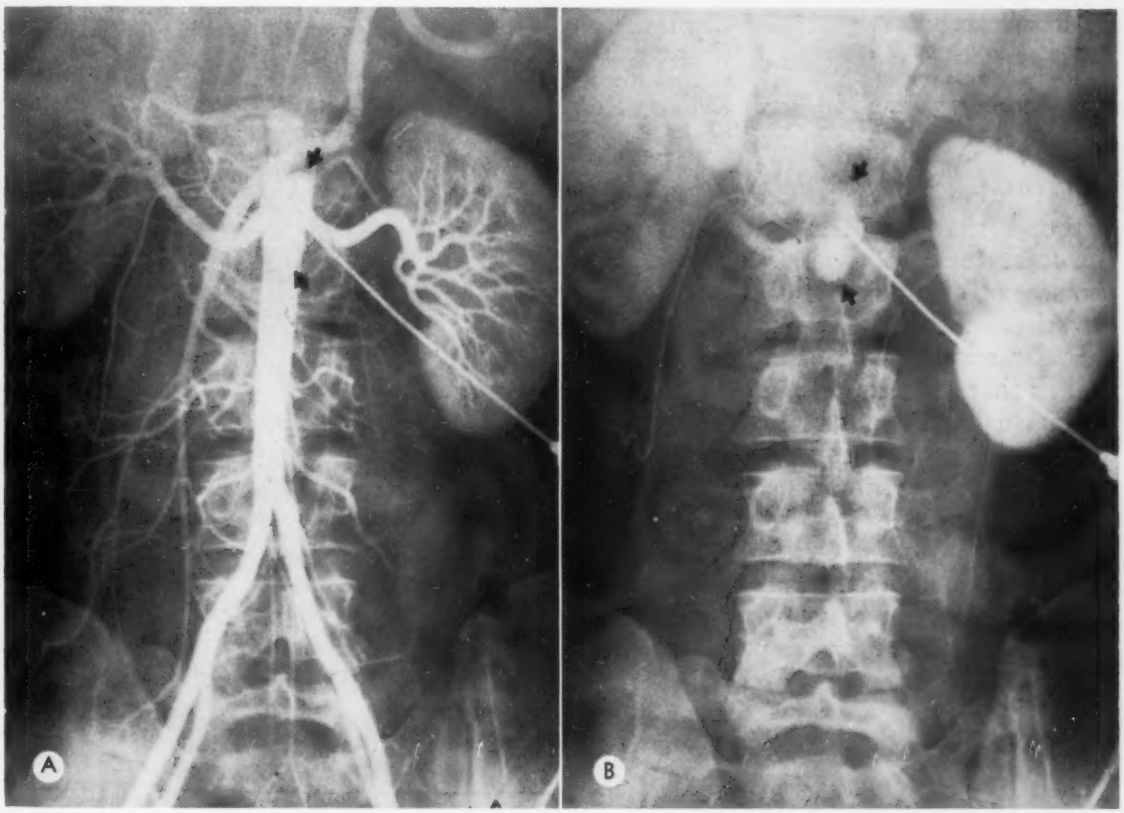

Fig. 4. (*A*) Good quality aortogram showing a short strategically located intramural injection at the level of the renal arteries. The volume injected at this level is 40 cc. Subsequently, 60 cc. was injected into the distal aorta to visualize the popliteal arteries. (*B*) Persistence of some of the intramural opaque material a few seconds following *A*. This thirty-seven year old man died of massive aspiration pneumonitis at a time when he was recovering slowly from uremia on the twenty-ninth day following aortography. No autopsy was performed.

Fig. 5. Aortogram showing uncomplicated partial intramural injection and demonstrating small saccular aneurysm of the right renal artery. The aneurysm was presumably related to the patient's hypertension, which was relieved following right nephrectomy.

well as through clinical observations, and it is proper that search should continue for the least toxic compound. However, one should not overlook the obvious insult to any tissue resulting from occlusion of its vascular supply. Our own experience, as well as that of Idborn,[5] has indicated that if a normal renal blood flow is maintained during the procedure serious permanent impairment of renal function does not necessarily result even with a fairly direct renal artery injection. Quite likely, as in our 3 fatal cases, ischemia and contrast material toxicity often play a combined role in producing tissue injury.

Our incidence of intramural injections has been determined entirely in retrospect; and had we not encountered serious complications from such injections, it is unlikely that their significance would ever have been fully appreciated. Furthermore, had the presence of an intramural injection been recognized on the test roentgenogram, additional injections would not have been made in the 2 patients showing large dissecting aneurysms at postmortem examination. We have found repeatedly that an intramural injection need not end so tragically if it is of reasonably small volume and not located near a vital vascular trunk. Unfortunately, even a small intramural injection may on rare occasions be of significance if it is located near an important aortic branch, as shown in Figure 4*A*. It is possible that many such injections have been unrecognized by others because their experience has not been as unfavorably unique as ours. It is also possible that complications have been produced in other series on this basis and attributed to other causes.

It seems evident that if the operator is unfamiliar with the roentgen signs of intramural contrast medium that the nature of the injury will be undetected in pertinent cases. This is especially true when one is dealing with a disastrous complication such as paraplegia, in which occasion may not arise to do surgery or to secure autopsy confirmation of the mechanism of injury. The same situation may exist when surgery is

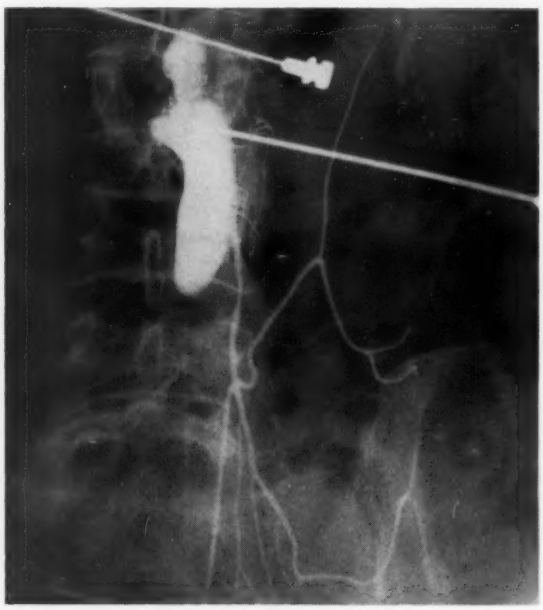

Fig. 6. Uncomplicated partial intramural injection. A successful aortography was performed a few minutes later from a higher puncture site, but we feel at this time that such a short time lapse is unduly hazardous.

required for an infarcted abdominal viscus, because, even though chemical injury is found, complete or partial occlusion of the vascular supply to the involved organ may have been responsible for the degree of sensitivity to the contrast medium shown. Accordingly, the roentgenographic appearance may provide the only, or at least the most definitive, part of the explanation for what has transpired in the often poorly explained complication of direct aortography.

In spite of the usual precautions taken by nearly everyone performing aortography, *i.e.*, use of a short bevel needle, puncture of the aorta only once, manual instead of high pressure mechanical injections, and avoidance of injection unless a good pulsatile flow of arterial blood issues from the needle hub with each systolic thrust, an intramural injection may occur. It is important therefore that any person performing direct aortography be thoroughly familiar with the roentgen appearance of intramural contrast medium. The procedure should be terminated if the test roentgenogram shows an intramural injection and the patient should be observed very closely with the possibility of surgical intervention in mind should a dangerous dissection have been produced by the time it is first recognized. The procedure should not be attempted again before at least twenty-four hours have elapsed. Delayed abdominal roentgenograms should be obtained within a few minutes to detect abnormal retention of iodinated compound by the kidneys or other viscera. If such retention is noted, one should anticipate need for whatever treatment may be feasible for that damaged organ.

Fig. 7. Test roentgenogram showing an uncomplicated partial intramural injection which was promptly recognized and the procedure was terminated.

SUMMARY

1. The roentgen signs of intramural contrast material are described. The importance of their recognition by everyone performing direct aortography is stressed.

2. In a series of 425 translumbar aortographies, intramural injection of varying amounts of the contrast material has occurred in 42 of the examinations. In 3 of these, death of the patient was either directly or indirectly related to the intramural injection, 2 of the patients dying with proved dissecting aneurysms. The other 39 patients had no significant complications.

3. The procedure of direct aortography should be discontinued and not attempted again for at least twenty-four hours if the test roentgenogram shows any evidence of an intramural injection.

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VOL. 81, NO. 5

NEW TECHNIQUE OF CONTRAST VISUALIZATION OF THE DISTAL AORTA, PELVIC AND LOWER EXTREMITY ARTERIAL SYSTEM IN OBLITERATIVE VASCULAR DISEASE*

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THE remarkable advances of peripheral vascular surgery in the past decade have depended greatly on the continued improvement of arteriographic techniques. The correlation of the clinical signs of insufficiency of the distal aorta and vessels of the lower extremities with the detailed arterial pattern is tantamount to successful management. Thus the choice of lumbar sympathectomy, endarteriectomy, grafting, or amputation is often no better than the accuracy of the preoperative information of the pathology involved.

Translumbar aortography has long been employed in the study of the abdominal aorta and its branches. The demonstration of the abdominal aorta and arteries of both lower extremities can be obtained by a single injection. Bilateral femoral arteriograms are also obtained in many institutions with or without the use of catheters. However, all these procedures have the shortcoming that serious proximal disease which is not yet clinically apparent may be overlooked. The Seldinger technique of percutaneous retrograde femoral catheterization of the aorta has not been widely accepted in this country. The possibility of shearing off the catheter, the occurrence of hematoma at the puncture site, the occasional dislodging of plaques with the catheter, and the inability to advance the catheter even above incomplete obstructions in obliterative vascular disease are the most common objections to its use. Aortography, therefore, is still considered as the best of the present day procedures,[3,4,5,9] although serious complications continue to occur despite meticulous technique and the smaller volume, lower concentration and less irritating effect of the contrast materials employed.[1,2,6,7]

In view of these limitations, in 1956 the use of percutaneous retrograde femoral arteriography was begun at the United States Naval Hospital, St. Albans, New York. This technique has been reported as valuable in obstetric and gynecologic problems[8] and has been found easy to perform and as safe as peripheral femoral arteriography.

PROCEDURE

After a clinical diagnosis of obliterative vascular disease of the lower abdominal aorta, iliac, or femoropopliteal arteries, an attempt is made to localize the major obstructed areas by corraliting the history and physical examination. Thus, a difference in contralateral pulses may be evident; a complete lower aortic obstruction may present as a typical Leriche syndrome; a diminished or absent femoral pulse, associated with cramping of the muscles of the buttocks or posterior thigh, may represent iliac occlusion; and calf pain, associated with good femoral pulsations, may indicate superficial femoral or popliteal arterial disease.

Under local anesthesia the femoral artery, 1 cm. inferior to the inguinal ligament on the side of least involvement, is percutaneously punctured with a 16 gauge Lindmann needle. The more severely affected side should not be utilized initially, since the circulation may be precarious and further insult to that arterial system can almost always be avoided with this technique. A tourniquet is then placed as

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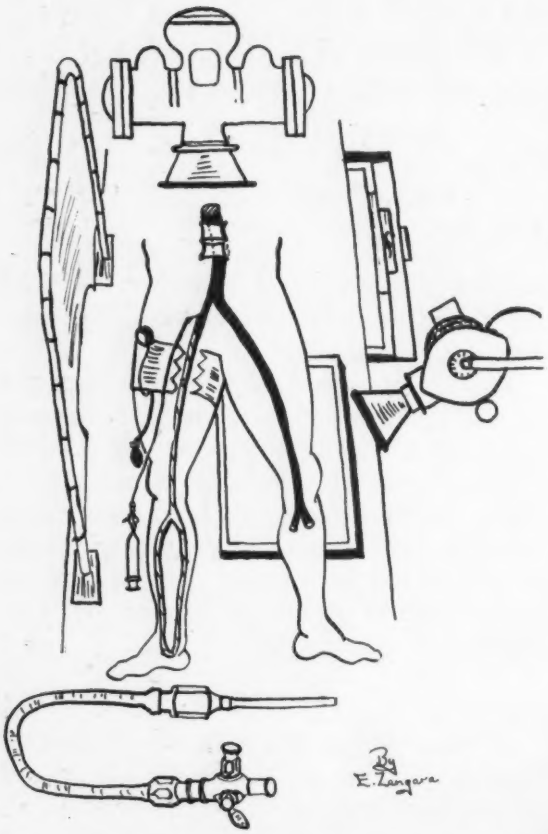

FIG. 1. The part of the arterial system indicated in solid black is visualized on the first injection with the tourniquet inflated. The tourniquet is then removed, and the part of the arterial tree indicated in broken lines is outlined on the second injection. It is especially important to visualize the outflow tract distal to an obstructed area.

cephalad as possible on the thigh of the side to be injected. The patient performs a Valsalva maneuver during the injection to slow the flow of blood. Thirty-five cc. of 50 per cent hypaque is injected by hand as rapidly as possible and a single exposure made of the lower abdominal aorta, iliac, and pelvic arteries. Serial roentgenograms at one second intervals are then taken of the femoral artery to the knee on the side of suspected pathology, which is the contralateral side of injection. While these roentgenograms are being developed, the ipsilateral extremity is injected after removal of the tourniquet. The arterial vascular pattern of the abdominal aorta, distal to the renals, iliac, and femoral arteries of both lower extremities as well as the vasculature of the leg on the injected side is thus obtained by these two injections (Fig. 1 and 2). This volume of contrast material makes general anesthesia unnecessary and patient cooperation has been satisfactory. If a complete block of the aorta is present, either femoral artery is injected using anatomic landmarks and the vasculature distal to the block evaluated. In this case translumbar aortography is performed with 10 cc. of 50 per cent hypaque to determine the proximal extent of the block.

In evaluating postoperative results the nonoperated side is again chosen to insure against injury to the graft. Serial roentgenograms are taken to study the vascular pattern in detail.

Figures 3 through 7 illustrate various vascular problems evaluated by this simple technique.

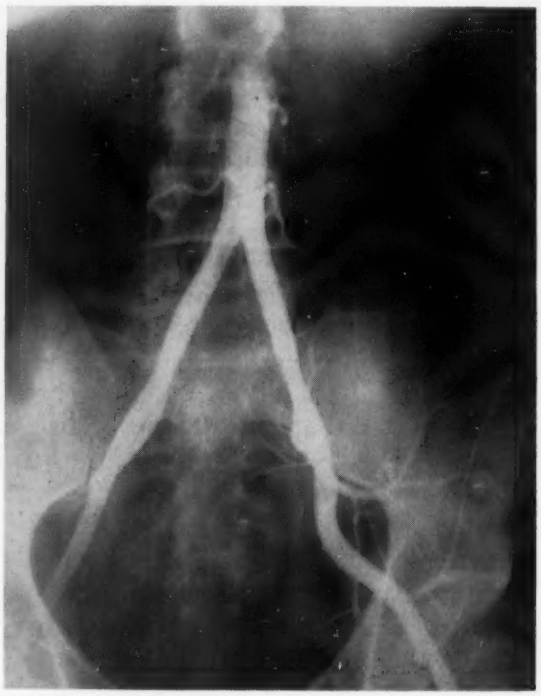

FIG. 2. Normal retrograde femoral arteriogram. During the injection the contrast material ascends to the level of the renal arteries, but not above this. The large flow of blood to the kidneys probably cannot be overcome without exerting unsafe pressures.

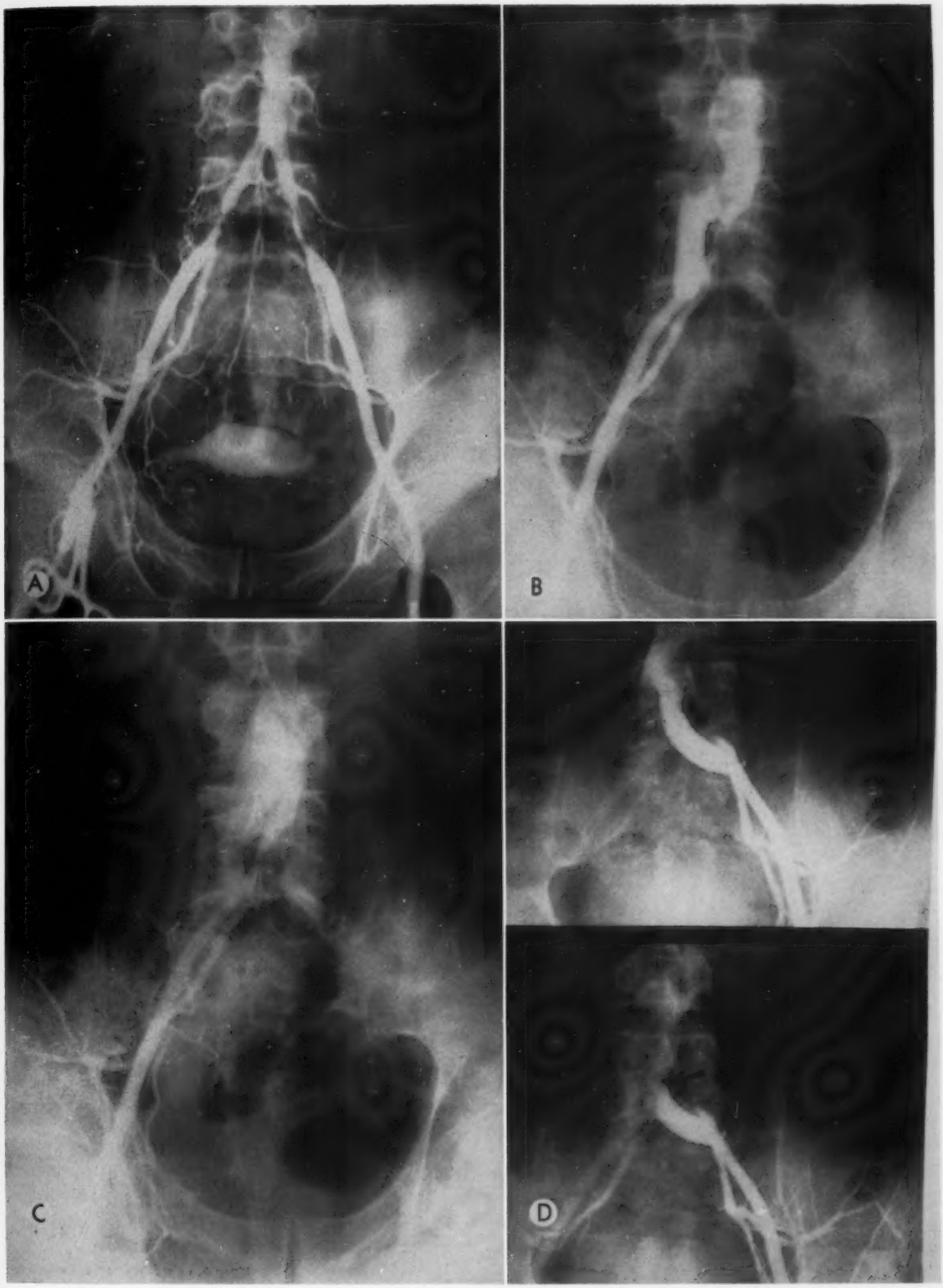

FIG. 3. (*A*) Diminished femoral pulsations are present bilaterally. The right retrograde femoral arteriogram demonstrates a plaque at the origin of the right common iliac artery and bilateral partial occlusion of the common iliac arteries over a distance of 2 cm. Note the normal arterial pattern and concentration of refluxed contrast material in the left superficial femoral and profunda femoral arteries. The lower extremity vessels were normal. A knitted bifurcation dacron graft was utilized to bypass the obstructions. (*B*) Postoperative retrograde femoral arteriogram demonstrates the patency of the right limb of the "Y" bifurcation graft. (*C*) Five-tenths of a second later the base of the graft which is anastomosed to the aorta is well visualized, but the left limb of the graft is poorly seen suggesting a thrombus or kink. (*D*) A left retrograde femoral study demonstrates the patency of the graft, but a minimal kink of the left limb is visualized. This explained the poorer left femoral pulse as compared to the excellent right femoral pulse. The bilateral intermittent claudication which was marked before surgery has completely disappeared.

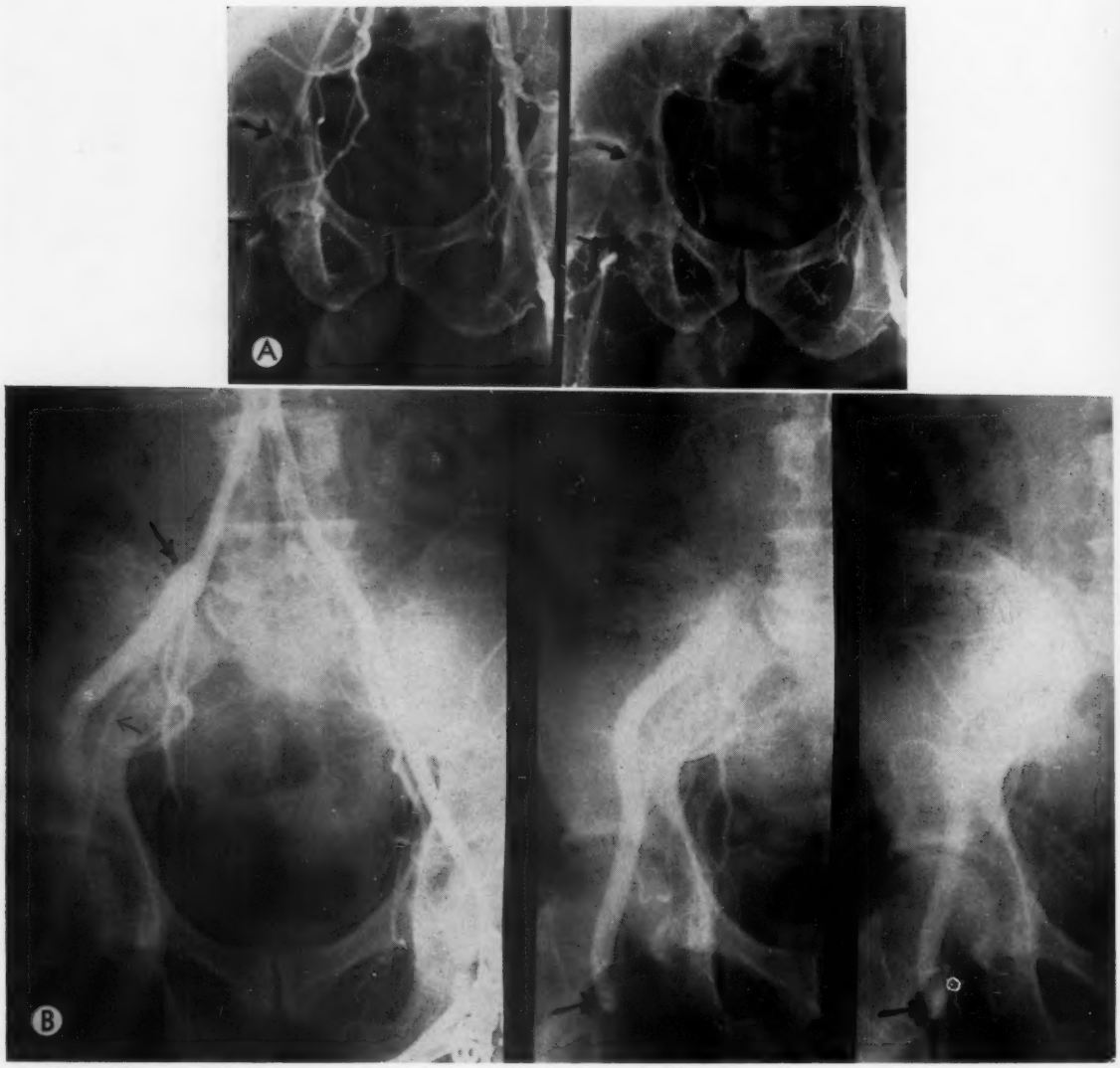

FIG. 4. (*A*) Because of intermittent claudication of the right thigh and leg, left retrograde femoral arteriography was done. This demonstrated a normal distal aorta, and the arteriogram on the *left* reveals an occlusion of the right external iliac artery (upper arrow). The arteriogram on the *right*, exposed two seconds later, shows the collateral circulation and extent of the block. Note the normal superficial femoral outflow tract. A woven dacron graft was used to bypass the obstructed segment. (*B*) One month postoperatively left retrograde femoral arteriograms revealed the graft extending from the right common iliac artery to the superficial femoral artery. The three exposures were made at one half second intervals. Note the absence of the previously visualized collateral circulation.

DISCUSSION

The technique of translumbar aortography has been available for the past twenty-five years. For the last fifteen years this route of investigation has been generally adopted in the United States, and has been found particularly valuable in renal, adrenal, retroperitoneal, and peripheral vascular pathology. Although extensively employed in the rapidly progressing field of vascular surgery on the aorta and its branches, complications continue to be reported. The incidence of these complications is rather small, but the permanent disability and death which occasionally occur indicate the need for a safer method of investigation. Since most of the occlusive diseases of the aorta and its branches occur distal to the origin of the renal arteries, retrograde femoral arteriog-

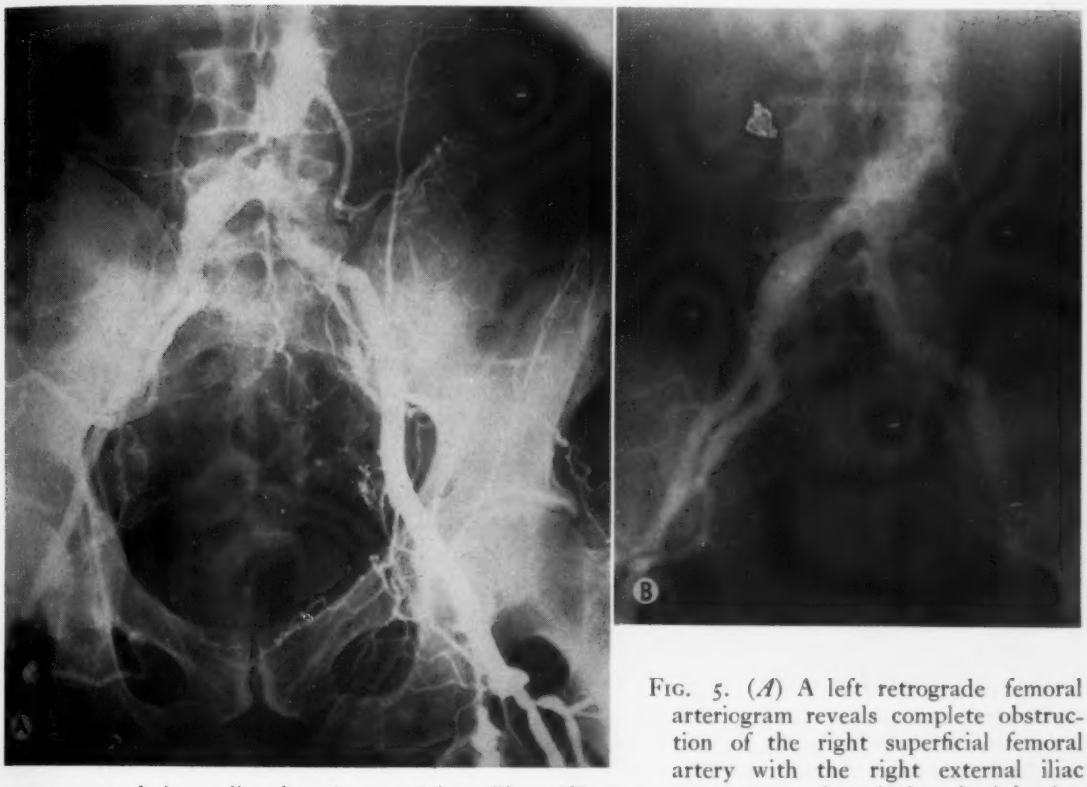

Fig. 5. (*A*) A left retrograde femoral arteriogram reveals complete obstruction of the right superficial femoral artery with the right external iliac artery smooth in outline but decreased in caliber. (*B*) A roentgenogram taken during the injection demonstrates the narrowing of the right common iliac artery at its origin with poststenotic dilatation of the remainder of the common iliac artery.

graphy should prove adequate in over 90 per cent of all lower aortic lesions and in all complete or partial occlusions distal to the bifurcation of the aorta. In such instances retrograde femoral arteriography should be employed first, since this safer procedure usually gives the complete information necessary for surgical correction.

In cases of complete aortic occlusion a twofold purpose is served. First, the status of the arteries peripheral to the block is determined. Secondly, the use of only 10 cc. of 50 per cent hypaque for translumbar aortography prevents an unnecessary and dangerously high concentration of contrast material in the renal, lumbar, mesenteric and ovarian arteries.

Fig. 6. Intermittent claudication of the left lower extremity is seen to be caused by compromise of the lumen of the distal aorta and left common iliac artery.

FIG. 7. Surface anatomy permits the retrograde femoral artery to be utilized in evaluating the vasculature distal to the aortic block. Note the small size of the external iliac artery and sudden increase in caliber at the insertion of the collateral afferent arteries. An aortogram demonstrated the upper level of the obstruction to be 1 cm. below the renal arteries.

Although a serial changer is of value in obtaining visualization of the contralateral vasculature, the routine roentgenographic equipment available in any hospital is adequate. A portable roentgenographic unit is satisfactory for the demonstration of the lower extremity vasculature.

The opacification produced by this procedure exceeds that seen following aortography with much more concentrated solutions. This is due to the complete replacement of the blood with refluxed contrast material, instead of the continuous dilution of the contrast column which occurs in aortography.

Hypaque has been found to be the most satisfactory contrast material because of the minimal oscillographic changes it causes in the injected extremity and the minimal patient reaction. For this reason, a local rather than general anesthesia is preferred.

CONCLUSION

The technique of percutaneous retrograde femoral arteriography has been presented as a simple and safe method of studying the lower abdominal aorta, iliac arteries, and the lower extremity arteries in vascular disease. The successful employment of this technique in 86 patients has been especially encouraging in view of the lack of significant morbidity.

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Vol. 81, No. 5

FUNCTIONAL ASCENDING PHLEBOGRAPHY OF THE LOWER EXTREMITY BY SERIAL LONG FILM TECHNIQUE*

EVALUATION OF ANATOMIC AND FUNCTIONAL DETAIL IN 62 EXTREMITIES

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THE superficial veins of the leg are, in general, readily accessible for clinical evaluation, but the deep veins are hidden within their muscular compartments. Phlebography will demonstrate the anatomic and pathologic status of these deep veins and can be helpful in establishing diagnosis and in guiding rational treatment of venous abnormalities.

However, it has been a difficult problem to develop a simple method for accurate deep phlebography which can provide both anatomic and functional information over the long area which must be studied.

Important technical difficulites which have had to be overcome include: means of introducing the opaque material into the deep veins; the development of safe but effective contrast materials which can be injected in quantities large enough to overcome dilution; proper means of delaying the emptying of contrast material to encourage its mixture with the blood in all deep veins; proper tests to differentiate normal from abnormal function; and the development of techniques providing good roentgenographic detail over the entire length of the leg.

Even when most of these difficulties have been overcome, the examiner is still faced with the problem of identifying veins which are variable in course and character, and are frequently overlapped on the roentgenogram. Consequently, phlebography has not proved popular among radiologists and other physicians who only occasionally are faced with a clinical problem of deep vein disease.

There have been continued advances in overcoming these difficulties since Berberich and Hirsch[4] in 1923 first described the feasibility of demonstrating the veins roentgenographically by injecting a radiopaque substance. Bauer[1] and also Dougherty and Homans[6] advocated the introduction of the contrast material into a foot vein to visualize the veins of the entire leg. Bauer[2] used a tourniquet at the ankle to assure passage of the contrast material into the deep system. Dow[7] proved that, as long as an obstructing bandage was placed above the site of injection, deep veins were adequately visualized whether the injection was made on the medial or lateral aspect of the foot.

Many different contrast materials have proved to be safe and effective. Of more importance is the quantity of contrast material which is used, as Felder and Murphy[9] demonstrated. A significantly greater number of veins was visualized when 40 cc. rather than 20 cc. of contrast material was injected.

Scott and Roach,[18] Greitz,[11] and others have demonstrated that placing the patient in a semierect position very adequately delays emptying of the veins. And the fact that all contrast materials have a specific gravity greater than that of blood serves to accentuate this delay and also results in improved visualization of the valves.

Greitz[11] suggested that active movement

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of the leg muscles, by having the patient raise himself on his toes, could increase the number of deep veins visualized. Shumacker *et al.*[19] and Bauer[3] presented small numbers of normal and abnormal extremities of which they took roentgenograms before and after similar exercise, but they did not establish extensive criteria for differentiating the normal from the abnormal functionally.

The problem of obtaining serial full length roentgenograms of the lower extremity has been solved by Rogoff,[17] Eyler,[8] and others.

In 62 extremities we have evaluated in detail a technique of functional phlebography similar to that described by Shumacker *et al.*[19] and Bauer.[3] By history and physical examination 13 extremities were considered normal, 10 extremities were considered to be postphlebitic, and 39 extremities were felt to exhibit only varicose veins. The practicality of performing such an examination has been evaluated; the frequency of visualization of the important deep veins of the legs has been determined; the anatomic appearances of normal and abnormal veins have been carefully observed; and the significance of this examination in determining the functional capacities of the deep veins has been studied.

METHOD

1. The patient was placed supine and semierect on a roentgenographic table which was raised to 60° from the horizontal plane.

2. Either a standard 18-gauge needle was introduced percutaneously or a small polyethylene catheter was directly introduced through a skin incision into any superficial vein on the dorsum of the foot. The needle or catheter was connected by a three-way stopcock to a bottle of normal saline solution and a continuous drip was started. A rubber tourniquet was then tightly placed around the ankle.

3. Following a negative skin test, 40 cc. of 50 per cent hypaque was injected into the foot vein over a period of one minute.

4. At the end of injection, the first exposure was made on a 14×34 inch long film.

5. The tourniquet was removed and the patient was asked to raise himself on his toes, forcibly, ten times.

6. A second long roentgenogram was then taken. The time lapse between the first and second roentgenogram was thirty-five to forty seconds.

7. In most instances, a third 14×17 inch roentgenogram centered under the pelvic region was taken about forty-five seconds after the second roentgenogram.

8. The patient was then returned to a horizontal position and the saline solution was rapidly flushed through the catheter or needle.

ROENTGENOGRAPHIC EQUIPMENT

A spring cocking 14×36 inch Potter-Bucky diaphragm is permanently mounted under the top of a tilting table. Fourteen by thirty-six inch cassettes are loaded with two 14×17 inch films placed end to end inside the cassettes. A target-film distance of approximately 56 inches is required to expose the entire 34 inch length. A wedge filter has been constructed for insertion at the tube window in order to equalize roentgenographic density from thigh to calf. A lead diaphragm is also placed at the tube window to act as a primary cone for the roentgen-ray beam. A 14 inch width is employed since both extremities are frequently examined simultaneously. Average technical factors at 56 inch target-film distance have been 75 kv., 200 ma., at 0.5 seconds, using Par-Speed screens and Blue Brand film. Cassettes are changed by hand without difficulty, since roentgenograms are taken at a leisurely pace. The general features of the equipment have been described by Rogoff.[17]

RESULTS

I. PROBLEMS AND COMPLICATIONS OF METHOD

The method proved to be practical and relatively simple to execute since the necessary roentgenographic equipment was al-

ready on hand. In general, superficial veins on the dorsum of the foot or ankle were readily available. Cut downs were usually necessary only when marked edema was present.

Most patients experienced a bursting discomfort in their foot during the injection of the opaque material, and some complained of pain in the calf. The pain rapidly disappeared after returning the patient to a horizontal position when saline solution was infused through the veins. There were no allergic reactions to the hypaque.

Six patients experienced syncope when being tilted from the horizontal to the semierect position even before the procedure was started, but after a brief rest they were able to complete the examination. Two patients vomited at the end of the procedure. There have been no instances of superficial or deep phlebitis following the procedure. Most examinations have been performed upon both extremities at the same time. Out-patients have been able to return home soon after the procedure.

II. Visualization of Important Deep Veins

It was possible to fill enough of the important deep veins of both the lower leg and thigh with contrast material to visualize adequately the anatomic features in almost all the extremities (Table I). The anterior tibial, posterior tibial, peroneal and superficial femoral veins were adequately visualized in more than 90 per cent of the extremities studied. When demonstrated, almost all of the peroneal and posterior tibial veins from the level of the tourniquet to their entrance into the popliteal vein were seen. However, frequently only the proximal few centimeters of the anterior tibial vein were filled. The popliteal vein was visualized in 97 per cent of the examinations. The deep femoral vein was least frequently seen but, when present, was almost completely depicted, probably because in all of these instances there was a distal communication between the deep femoral vein and either the superficial femoral or popliteal vein (a "circle").

Table I

Percentage of Extremities in Which the Important Deep Veins of the Leg Were Visualized in 62 Ascending Phlebograms

	Per Cent		Per Cent
Anterior Tibial	90	*Sural*	67
Part	23		
All	67		
		Popliteal	97
Peroneal	90		
Part	3		
All	87	*Superficial Femoral*	90
Posterior Tibial	94		
Part	2	*Deep Femoral*	61
All	92	Part	8
		All	53

In studying these phlebograms it was obvious that by having serial roentgenograms we not only saw deep veins more frequently but also more clearly. For example, the veins of the thigh were frequently more adequately visualized on the second roentgenogram, which often rendered conclusive detail in calf veins as well.

III. Anatomic Characteristics of the Veins

A. *Veins of the Lower Leg* (Table II).

1. *Normals* (Fig. 1). The anterior tibial, peroneal and posterior tibial veins are the main deep venous channels in the lower leg. In the normal extremities, the hypaque was generally confined to these veins. The veins were frequently double. They presented a uniform tapering outline and their greatest width varied between 0.4 and 0.9 cm. Several valves were always present in these normal veins. The sural veins, which are smaller veins draining the gastrocnemius muscle and which usually enter the popliteal vein directly, were prominent in most normal extremities. Short segments of perforators ending at a competent valve without filling the superficial veins were also frequently seen. Since we had not used stereoscopic or lateral views, we were unable to determine whether many veins were

TABLE II

ANATOMIC CHARACTERISTICS OF LOWER LEG VEINS

	Normal Main Veins	Abnormal Main Veins	Surals Present	Competent Perforators Present	Communicators and Collaterals		
					None	Few	Numerous
Normal Extremities (13)	12	1	10	9	3	9	1
Postphlebitic Extremities (10)	1	9	2	1	0	1	9
Extremities with Varicose Veins (39)	25	14	27	25	3	10	26

deep or superficial. Therefore, veins which could not be identified as main deep venous channels, sural veins, or competent perforators have been arbitrarily classified as "communicators" if they passed predominantly in a horizontal direction and "collaterals" if they ran in a vertical direction. Many of the communicators are probably incompetent or competent perforators, or merely communicating veins among superficial channels. Most of the collaterals are probably superficial veins but some may be deep collaterals. In normal extremities there are very few communicators and collaterals visualized.

2. *Postphlebitics* (Fig. 2 and Fig. 3). The anterior tibial, posterior tibial and peroneal veins occasionally appeared normal in postphlebitic extremities. If abnormal, they had irregular walls, were valveless, and were at times tortuous. Of particular significance to us was the fact that these veins were not dilated. Their greatest width varied only from 0.4 to 0.9 cm. Sural veins were not seen as frequently, probably because they were now valveless and could not be differentiated from other collateral veins. Competent perforators were also rare. However, there were numerous valveless and frequently tortuous communicators and collaterals.

3. *Varicose veins* (Fig. 4). In general, the main deep venous channels of extremities with varicose veins appeared normal. There was a slight tendency for normal appearing veins to be wider than those in normal extremities. Also, abnormal veins were frequently seen, showing localized fusiform dilatation and an absence of valves. However, the veins above and below these "deep varicosities," in general, appear normal. Sural veins and competent perforators were seen but not as frequently as in the normal. There was a definite increase in communicators and collaterals in these varicose extremities.

B. *Veins of the Thigh* (Table III).

The popliteal, superficial femoral and

TABLE III

ANATOMIC CHARACTERISTICS OF THIGH VEINS

	Normal Main Veins	Abnormal Main Veins	Communicators and Collaterals		
			None	Few	Numerous
Normal Extremities (13)	13	0	10	3	0
Postphlebitic Extremities (10)	0	10	0	5	5
Extremities with Varicose Veins (39)	24	15	16	16	7

Fig. 1. Normal extremity. *Left:* The proximal third of the anterior tibial (A. T.) vein and all of the peroneal (Per.) and posterior tibial (P. T.) veins are visualized. The popliteal (Popl.) and S. femoral (S. Fem.) are smooth-walled and contain valves. There is a distal communication between the S. femoral and D. femoral vein in this case. Paired competent perforators (Perf.) and sural veins are present in the calf. *Right:* Postexercise the contrast material has been emptied from the veins except for a small amount in the valve cusps.

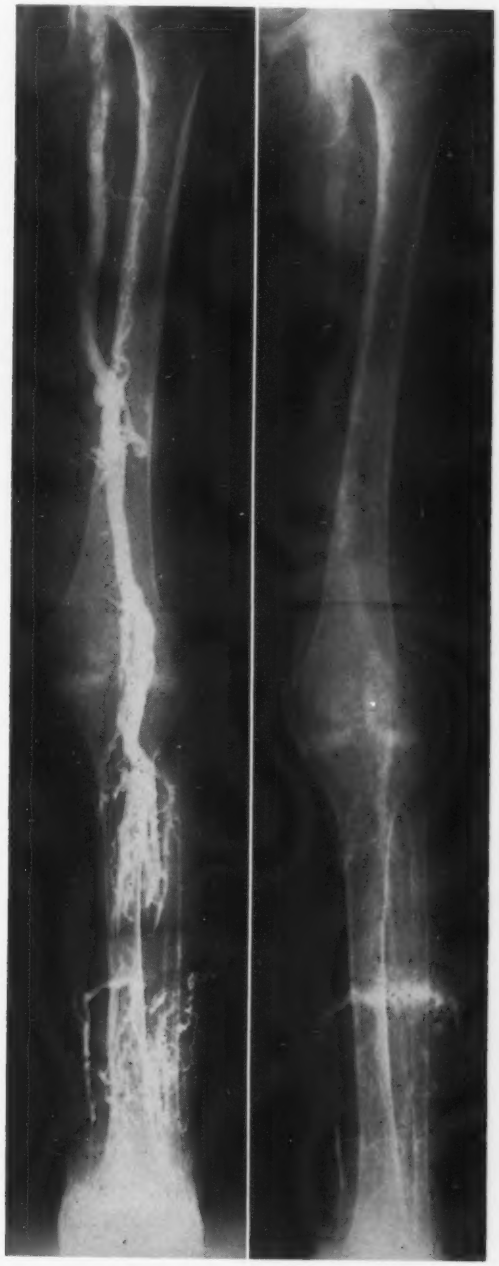

Fig. 2. Postphlebitic extremity. *Left:* Most of the deep calf veins appear normal and there are definite valves. The popliteal and S. femoral vein have irregular walls and no valves are seen, characteristic of a completely recanalized vein. *Right:* After exercise contrast material is still present in the deep veins and increased numbers of communicators and collaterals are visualized. The tourniquet was placed higher than usual because of the presence of an ulcer, and contrast material entered the smaller vessels after the tourniquet was removed.

FIG. 3. Postphlebitic extremity. The normal deep veins cannot be identified but are replaced by communicators, collaterals, and probably partially recanalized veins. The distal greater saphenous vein (Gr. S.) appears normal. (a) This opacified channel is along the course of the S. femoral vein and may represent recanalization, collateral or venae comitantes.

FIG. 4. Varicose veins. *Left:* This is the appearance of the deep veins in some extremities with superficial varicose veins. There is fusiform dilatation of the peroneal and posterior tibial veins with no valves seen in the dilated portion (*i.e.*, deep varicosities). The popliteal (a) and S. femoral vein (b) are smooth-walled, and without valves. *Right:* After exercise there has been poor emptying of the deep veins of the calf and an increased number of communicators and collaterals (c) are visualized.

deep femoral veins are the main deep venous channels visualized in the thigh. Since the superficial femoral vein is not a superficial vein but is a deep vein, we call the superficial femoral vein "S. femoral" and the deep femoral "D. femoral."

1. *Normals* (Fig. 1). In normal extremities the popliteal, S. femoral and D. femoral veins were smooth-walled and of uniform caliber except for some minimal dilatation at the site of valves. The greatest width of the popliteal vein varied from 0.9 to 1.2 cm. and 1 or 2 valves were seen. The greatest width of the S. femoral veins varied from 1.1 to 1.6 cm. and from 1 to 6 valves were demonstrated. In these cases, 28 per cent of the S. femoral veins were bifid. The D. femoral vein was not always visualized. Whenever most of this vein was seen, a distal communication between either the popliteal or S. femoral vein and the D. femoral vein was noted. Only a few communicators and collaterals were observed in normal extremities.

2. *Postphlebitics* (Fig. 2 and Fig. 3). The popliteal and S. femoral vein appeared abnormal in all of the patients with postphlebitic extremities. Some veins exhibited complete recanalization in which case the walls were irregular and valveless. However, these recanalized veins were *not dilated.* In other cases, there were only thin tortuous lines of opacity seen along the course of the S. femoral vein, which had the appearance of venae comitantes or collaterals ascending on either side of the obliterated deep vein.[5] These channels were also valveless. An increased number of communicators and collaterals were seen in the thigh as well as the calf in these extremities.

3. *Varicose veins* (Fig. 4). In patients with varicose veins, the majority of the main deep veins of the thigh appeared normal. However, many were abnormal. These abnormal veins, in general, exhibited smooth walls without valves and were usually dilated. Dilatation and smooth walls differentiated them from the valveless irregular, recanalized veins of the postphlebitic extremity. Two veins were also considered abnormal which exhibited very marked dilatation at the site of apparent valves. There was also an increased number of communicators and collaterals in these extremities.

IV. Functional Characteristics of Normal and Abnormal Deep Veins

The complete functional examination was performed on 52 extremities of which 13 were normal, 6 were postphlebitic, and 33 exhibited varicose veins. In all of these examinations, following exercise, there was evidence of cephalad movement of the opaque medium.

In normal extremities, this movement generally left only small amounts of opacity in the calf veins, particularly in the valve cusps. There was an accompanying decrease in the concentration of the opaque medium in the thigh veins. There was usually a decrease in the number of communicators and collaterals (Fig. 1).

In postphlebitic extremities there was little change in the concentration of the opaque medium of the main deep veins of the calf after exercise, the thigh veins showed increased opacification, and the collaterals and communicators of the calf usually showed increased filling (Fig. 2).

In studying the extremities with varicose veins, no over-all postexercise pattern was at first discernible. As our study progressed, however, it was first noticed that the main anatomic difference between the normal and postphlebitic extremities was that all of the latter had irregular, valveless deep thigh veins of normal width, and an increased number of collaterals and communicators. The varicose vein cases, on the other hand, tended to show smooth valveless and slightly dilated deep systems, also with increased collaterals and communicators. Therefore, another more careful analysis was made of the functional response in all extremities, taking these anatomic differences into consideration.

The decrease in opacification (emptying) of the deep calf veins was graded as poor,

FIG. 5. Communicators and collaterals. (*a*) Normal deep calf veins with no definite communicators (Comm.) and collaterals (Coll.). (*b*) Some dilated deep calf veins and a few communicators and collaterals. (*c*) The deep calf veins have irregular walls and no valves, characteristic of recanalized veins, and there are numerous communicators and collaterals.

good or excellent (Fig. 6 and 7). Poor emptying signified that there was little or no change in the opacification of the deep calf veins following exercise. Good emptying signified that only small portions of the veins and valvular cusps remained opacified. Excellent emptying signified that only the valve cusps retained the contrast material. The degree of emptying was then related to the status of the deep thigh veins and to the number of communicators and collaterals in the calf (Table IV and Fig. 5).

In the presence of normal thigh veins with only a few communicators and collaterals, the vast majority of the deep veins of the calf showed good to excellent emptying. However, if there were more numerous communicators and collaterals, the emptying was more apt to be poor or good. In the presence of abnormal thigh veins, emptying of the deep calf veins was never excellent and usually poor regardless of the number of communicators and collaterals.

The postexercise changes in the appearance of the communicators and collaterals were also related to the status of the deep veins and to the number of communicators and collaterals (Table V). If only a *few* communicators and collaterals were visualized in the presence of *normal* deep thigh veins, they tended to empty after exercise. But in the presence of *abnormal* thigh veins, such few communicators and collaterals were more likely to fill or increase in number after exercise. When the examination

Fig. 6. Good functional response. *Right:* Postexercise only a small amount of contrast material is present in the deep veins and valvular cusps. There has been good emptying. The few communicators and collaterals seen before exercise (*left*) are not opacified and are empty postexercise.

showed numerous collaterals and communicators, these tended to fill or increase their number after exercise, whether the deep veins were normal or abnormal (Fig. 7).

DISCUSSION

The method of functional ascending phlebography described here has proved to be a very practical one in our hands. The use of a foot vein for injection followed experience with a technique of lesser saphenous phlebography, which routinely required an incision for the cannulation of the vein.[5] Percutaneous placement of the needle into a foot vein is a much more desirable procedure. Hypaque was selected as the contrast medium because of its availability in our radiologic department, and we have been impressed with the low rate of complications from its use. Hypaque, like most contrast media, has a specific gravity greater than that of blood, which desirably decreases its rate of emptying. The use of 40 cc. of this substance, rather than smaller amounts, has certainly increased the number of veins which we have been able to demonstrate.

The application of a tight tourniquet at ankle level to increase the filling of the deep veins has, in general, been successful. However, it may be responsible for the fact that

TABLE IV

EMPTYING OF THE DEEP CALF VEINS RELATED TO THE STATUS OF DEEP THIGH VEINS AND TO NUMBER OF COMMUNICATORS AND COLLATERALS

	Few Communicators and Collaterals			Numerous Communicators and Collaterals		
	Emptying of Deep Calf Veins			Emptying of Deep Calf Veins		
	Poor	Good	Excellent	Poor	Good	Excellent
Normal Thigh Veins	3	11	8	3	5	2
Abnormal Thigh Veins	2	1	0	10	5	0

FIG. 7. Poor functional response. *Right:* Postexercise there is still heavy opacification of the deep calf veins and emptying is poor as compared to pre-exercise (*left*). There is increased filling of the communicators and collaterals postexercise.

TABLE V

EMPTYING OF COMMUNICATORS AND COLLATERALS RELATED TO STATUS OF DEEP THIGH VEINS AND TO NUMBER OF COMMUNICATORS AND COLLATERALS

	Few Communicators and Collaterals		Numerous Communicators and Collaterals	
	Empty	Filled	Empty	Filled
Normal Thigh Veins	13	3	2	8
Abnormal Thigh Veins	1	2	1	16

none or only part of the anterior tibial vein was visualized in 33 per cent of the extremities studied, since the tourniquet may obstruct the vein at the ankle level.[12]

A crucial point of technique is the use of the semierect as opposed to the horizontal position. Gravity delays emptying of the veins, allowing better mixing of the contrast material with the blood, and has improved the visualization of the valves (Fig. 8).[11,14]

It was obvious that the use of a standard exercise resulted in the redistribution of the contrast material, particularly its propulsion into the thigh and, therefore, increased the percentage of the veins visualized. This improved visualization is very apparent when our results are compared with those of Felder and Murphy,[9] who used a similar technique except that they did not have their patients exercise.

We have encountered some difficulty in identifying the deep calf veins when they were abnormal or overlapped by a profusion of communicators and collaterals. There is little doubt that the addition of stereoscopy or lateral views would help to overcome this problem. Yet, we feel that these additional roentgenograms would tend to complicate what has proved to be a simple procedure in our hands and would not proportionately improve the value of the method.

The anatomic characteristics of normal main veins as seen on ascending vertical phlebograms have previously been documented by Greitz,[12] Højensgaard,[14] and others, and our findings have been generally in agreement with theirs. Normal deep

FIG. 8. Value of semierect position. Phlebogram of the same extremity taken in the horizontal (*left*) and semierect (*right*) position. The roentgenogram in the horizontal position suggests segmental obstruction of the deep veins of the calf and popliteal vein. These veins are seen to be normal when the examination is performed in the semierect position.

veins have uniform parallel walls and exhibit valves. The use of a relatively large quantity of opaque material which is heavier than blood, the performance of the examination in a semierect position and the improved visualization offered by exercise have made it possible for us to definitely outline valves in all of the major deep veins in the 13 normal extremities which were studied.

Following an acute thrombosis, veins may completely recanalize or may remain almost, or completely, obliterated by organized thrombus.[15] It is generally accepted that a recanalized vein has irregular walls on phlebographic visualization.[1,6,7,9,13,14] The absence of valves in recanalized veins has been previously emphasized.[13,14] Although Højensgaard[14] felt that recanalized veins were sometimes dilated, this has not been mentioned by Greitz[13] or Murphy and Felder[16] as being a characteristic finding. Certainly, in this and previous studies,[5] we have not observed dilatation in veins which have been known to be postthrombotic.

In postphlebitic extremities main venous channels frequently cannot be identified but are replaced by thinner, tortuous, dilated veins. These channels are undoubtedly either dilated collateral veins such as Murphy and Felder[16] have described as "venous by-passes," or, in some instances, may be small channels through the main vein itself.[15] Occasionally, two collateral channels running parallel to the accepted course of the S. femoral vein in the anteroposterior view are seen. It seems likely that these collaterals are dilated venae comitantes.[5]

Most patients with varicose veins exhibited normal main deep veins. We were not impressed by the decrease in the number of valves in these normal appearing veins as was suggested by Højensgaard.[14] The segmental dilatation of the anterior tibial, posterior tibial or peroneal veins in some of the extremities was considered a normal variation by Greitz.[13] However, we have not observed this variation in normal or postphlebitic extremities and would suggest that these valveless segmental areas of dilatation are in reality deep varicosities. The S. femoral and popliteal vein have frequently appeared dilated and valveless. These changes have also been described by Bauer,[3] using a retrograde technique of phlebography, and by Ferreira *et al.*,[10] and ourselves[5] using a lesser saphenous technique of phlebography.

Only a few communicators and collaterals were visualized in normal extremities. Greitz,[13] Højensgaard,[14] Felder and Murphy[9] and others have described increased demonstration of such veins in postphlebitic extremities or in patients with varicose veins.

Bauer[1] reported a method of functional ascending phlebography to evaluate patients before and after popliteal vein ligation. His patients were placed in a supine position with the table tilted to 70° from the horizontal plane. He injected only 20 cc. of contrast material into a superficial vein on the dorsum of the foot and concentrated on the veins of the lower leg. His standard exercise consisted of "tramping down" on a pedal three times. He illustrated one normal extremity in which there was complete emptying of the deep veins of the lower leg following exercise. He also demonstrated a postphlebitic extremity which showed no emptying of the deep veins or communicators and collaterals with the exercise. Shumacker and coworkers[19] used a similar method to the one we have described, except that only 30 cc. of contrast material was injected and apparently no tourniquet was applied. In either one or two normal subjects, he described proximal propulsion of the contrast medium in both the deep and superficial venous systems after exercise. In extremities with varicose veins and in postphlebitic extremities, there was evidently still proximal propulsion of the contrast material in the deep veins, but the superficial and collateral veins showed poor

emptying. The number of extremities studied by this particular method or a detailed analysis were not presented.

When a contrast material is injected into a vein, it mixes with the venous blood and moves with it. Since the opaque substance is heavier than blood, gravity will exert some influence on this movement. In normal extremities the contrast material rapidly enters the deep veins and after the standard exercise we have used, it generally has been propelled proximally very promptly. Only small amounts of opaque material are seen in superficial veins. But in extremities in which the femoral and popliteal veins are abnormal because of the absence of valves due either to postphlebitic changes, congenital absences, or degenerative changes,[5] the opaque material does not empty as well from the deep veins of the lower leg. The propelling force of the exercise has been the same as in normal extremities, but the ineffective valvular system has delayed emptying or, in other words, has contributed to venous stasis. These abnormal extremities have numerous collaterals or communicators associated with primary varicose veins or their development following an acute deep phlebitis. The contrast material generally does not rapidly enter the deep venous system, and following exercise the number of minor veins visualized usually increases. Once again, there has been delayed emptying of the opaque material, or blood, which has contributed to venous stasis.

SUMMARY

1. A practical method is described for obtaining functional ascending phlebograms of the deep veins of the entire lower extremity. Important features of the technique are: (a) semierect position of the patient; (b) injection of at least 40 cc. of 50 per cent hypaque into any superficial vein on the dorsum of the foot with a tourniquet applied at the ankle; and (c) exposure of 34 inch long films of the leg before and after vigorous standard exercise.

2. Anatomic and functional information is reported after a detailed study comprising 62 extremities: 13 normals, 10 postphlebitics, and 39 with varicose veins.

3. In the vast majority of cases, the major deep veins of the calf and thigh have been demonstrated successfully by one injection and one examination.

4. The roentgenographic characteristics of normal and abnormal deep veins are described.

5. The movement of opaque material after exercise has been correlated with the presence or absence of valves in the deep thigh veins and with the number of communicators and collaterals shown in the lower leg.

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ADDENDUM

Since this manuscript was submitted, the authors have also discussed the "Clinical uses of functional ascending phlebography," *Angiology*, 1958, 9, 268–278.

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DEMONSTRATION OF LIVER REGENERATION BY SPLENOPORTAL VENOGRAPHY*

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THE entire right lobe of the human liver is being resected with increasing frequency for such indications as primary malignant tumors (hepatoma, cholangiocarcinoma and somatic sarcoma), unilobar metastases, cancer of the gallbladder, and occasional benign tumors. The right lobe consists of 80 per cent of the weight of the human liver, yet it now is feasible and relatively safe to perform total right hepatic lobectomy by the controlled hemostatic procedure of preliminary ligation and severance of the right hepatic duct, the right hepatic artery, and the right branch of the portal vein prior to transection of the interlobar septum. A residual 20 per cent of the human liver substance is enough for the maintenance of life.

The progressively increasing bulk of the left lobe is not due simply to vascular engorgement and edema, but rather to a true proliferation of new liver tissue. Once the immediate adaptation to the increased blood flow has occurred, chemical analysis of aliquot parts of the regenerated, as compared with the normal, liver show identical values for lipid, nitrogen and water content. Nuclear counts of homogenized liver tissue reveal a normal level in the rat within three days, increased above the normal by the fifth postoperative day, and then resumption of normal values. Similar studies of the residual dog liver confirm the tenet that the increased liver substance is due to a true regenerative hyperplasia of parenchyma and not a vascular artefact.

The rapidity and completeness of liver regeneration after subtotal hepatectomy appears to be inversely proportional to the size of the animal, *e.g.*, as in rats, dogs and humans. In rats the liver has completely regenerated within twelve to eighteen days after major lobectomies. In dogs the remnant of residual liver after 70 per cent hepatectomy has grown to the original proportion within eight weeks after the operation. The larger the proportion of the liver excised, the greater is the rapidity and the more complete the regeneration experienced; within the limits of survival it would seem proper to make the general statement that the stimulus to hepatic regeneration is directly proportionate to the percentage of liver excised.

The influence of the portal venous pressure on the start and completion of liver regeneration is important because this regeneration is intimately linked with the quantity of portal blood traversing the organ. After total right hepatic lobectomy and the consequent ligation of the right branch of the portal vein, portal hypertension in the remaining left lobe due to complete shunting of the portal stream is not only inevitable but highly beneficial in promoting rapid regenerative hyperplasia. The rapidity of regenerative hyperplasia of the residual liver tissue, other conditions being on a par, is directly proportionate to the degree of portal hypertension. The portal venous pressure returns to normal when the liver is anatomically and physiologically restored

In addition to the studies summarized in the foregoing statements, an excellent manner of demonstrating the rapidity and completeness of regeneration after major hepatectomy is by serial splenoportal venography. The value of this technique is summarized in this brief report.

METHOD AND MATERIAL

Splenoportal venography may be used to demonstrate the rapidity and completeness

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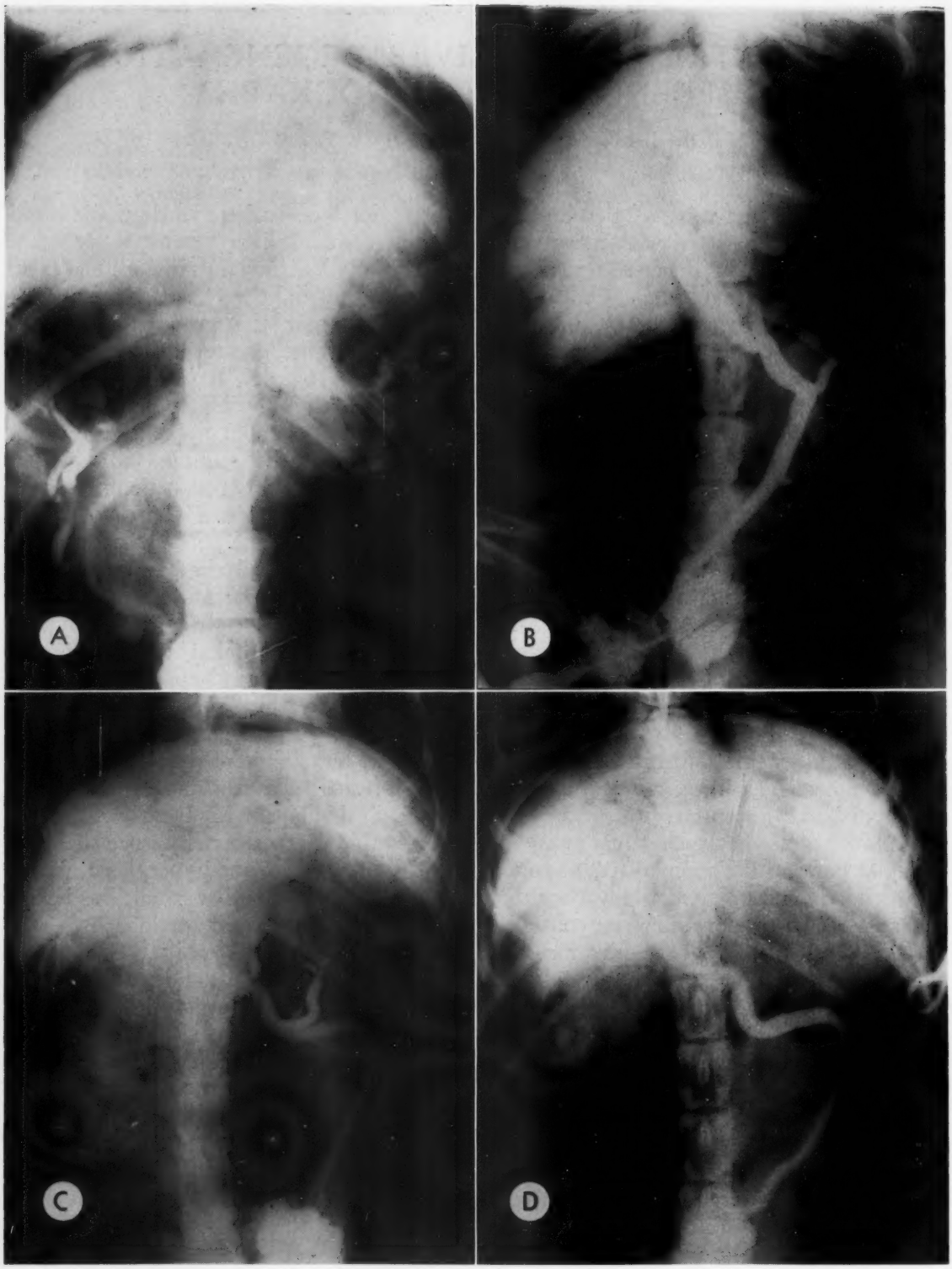

FIG. 1. (*A*) Splenoportogram shows the intact liver of the dog. (*B*) Splenoportogram immediately after 70 per cent hepatic lobectomy reveals the size of the remaining liver and the dilatation of the portal vein with some back pressure. (*C*) Splenoportogram two weeks later in the same animal demonstrates the increase in size of the remaining lobes with some venous engorgement. (*D*) Splenoportogram four weeks after partial hepatectomy in the same dog reveals that the mass of the liver has increased to almost the original size of the organ before partial hepatectomy.

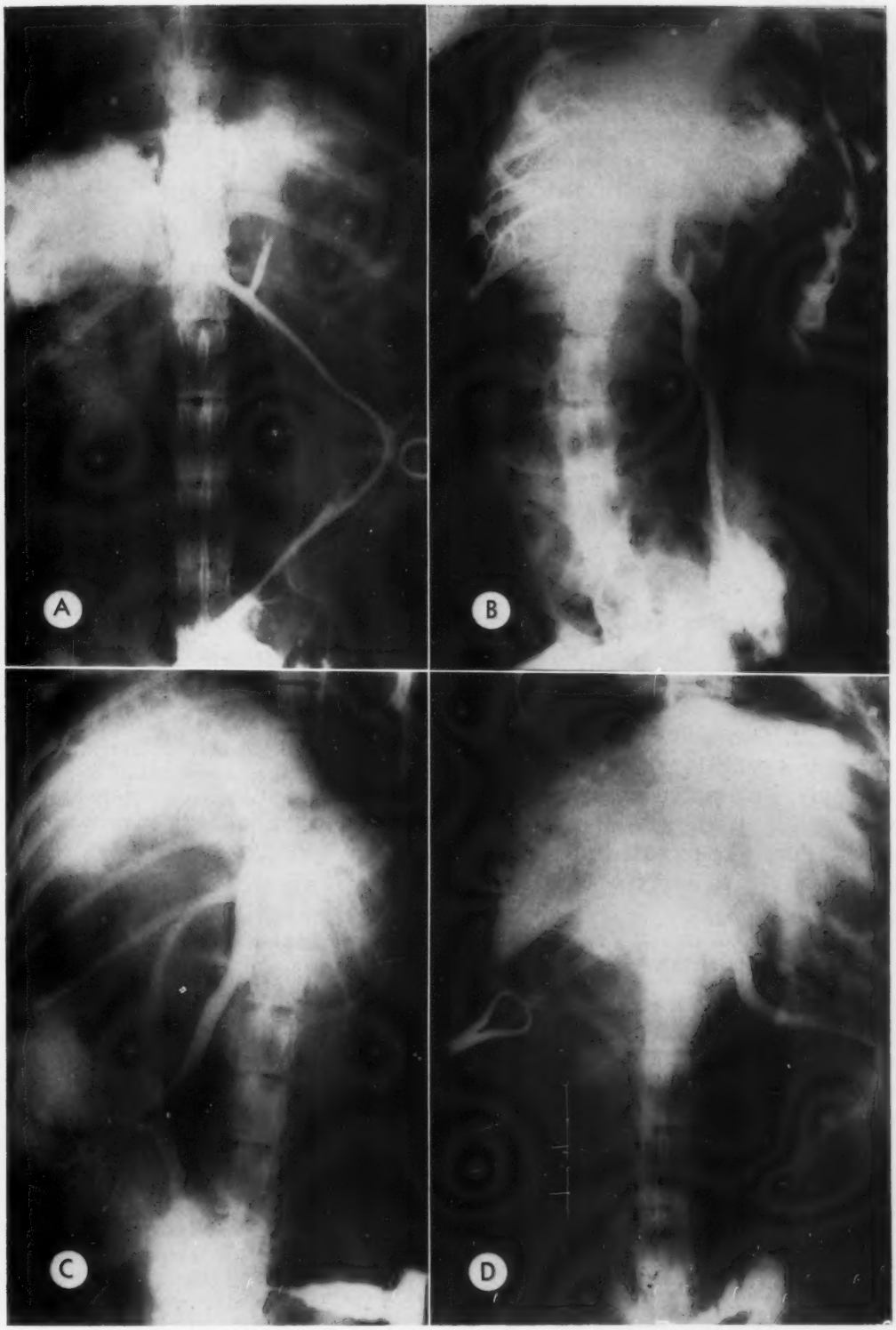

FIG. 2. (*A*) Splenoportogram shows the remaining single lobe immediately after partial hepatectomy. (*B*) Splenoportogram demonstrates the increase in liver mass two weeks after partial hepatectomy. Also, note the engorgement and dilatation of the portal tree (compare with Figure 1*C* which was taken two weeks after partial hepatectomy). (*C*) Splenoportogram indicates the enlargement of the liver mass four weeks after partial hepatectomy. (*D*) Splenoportogram reveals the enlargement of the liver mass six weeks after partial hepatectomy in the same dog.

of liver regeneration after partial hepatectomy. The experimental work was performed on dogs. The abdominal cavity was opened and the spleen located and brought into the wound. Diodrast of 70 per cent strength was used as the radiopaque material, giving 1 cc. of diodrast per kilogram of body weight. The diodrast was injected into the pulp of the spleen with an 18 gauge needle; before the completion of the injection a venogram was taken. This film was used as a control. A partial hepatectomy was performed in the manner previously described. As soon as the liver was removed, another splenoportal venogram was taken which furnished a comparative demonstration of the size of the remaining hepatic lobe.

Because the spleen of the dog is very movable and has no fixed point to introduce the needle directly through the skin for later procedures, the spleen was wrapped with omentum and sutured to the left abdominal wall. Two weeks later splenoportal venography was again performed on these dogs simply by transcutaneous puncture into the spleen at the site of its fixation. This procedure was repeated at two week intervals for six to eight weeks (Fig. 1, *A–D* and 2, *A–D*).

SUMMARY

Rapid increase in the size of the remaining lobe of the liver was noted, and is demonstrated in the accompanying illustrations.

As can be seen in the serial splenoportograms, the remaining liver regenerates rapidly, and in dogs the remaining lobe enlarges to the original normal size within four weeks after hepatectomy.

Extensive resections of the liver as a result of trauma, primary malignant tumors and metastatic or secondary cancers can be studied by this procedure.

It is also a diagnostic aid in obscure hepatomegaly, portal cirrhosis and disturbances in hepatic circulation.

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THE EFFECT OF IONIZING RADIATIONS ON CAPILLARY SPROUTING AND VASCULAR REMODELLING IN THE REGENERATING REPAIR BLASTEMA OBSERVED IN THE RABBIT EAR CHAMBER*

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THE REGENERATION of small blood vessels is a fundamental aspect of repair in tissues following injury. Granulation tissue, perhaps more accurately termed the repair blastema, is essentially a complex formation of regenerating capillaries. Agents which interfere with normal capillary sprouting in repair after injury usually arrest healing and restitution of continuity in tissues. This is due to the limited parenchymal regeneration which can take place in many organs. Furthermore effective parenchymal regeneration is dependent on regeneration of the stroma.

If the surviving tissues of the injured part have been exposed to ionizing radiations, or the actual zone of tissue destruction has resulted from such irradiation, healing may be greatly modified. In a previous paper,[14] a quantitative study of the effects of gamma rays on the growth of the repair blastema, observed in a Sandison-Clark type of ear chamber, was reported. The present paper deals with the microscopic appearances of regenerating capillary sprouts, observed *in vivo*, in rabbit ear chambers when subjected to single doses of roentgen or gamma irradiation. Descriptions of changes in capillary sprout formation will be given and related to the dosage employed. Quantitative observations previously made have shown that a single dose of the order 2,000 r is threshold for inhibiting growth of capillaries in most rabbit ear chambers and that further effective capillary sprouting is prevented by the development of a fibrous barrier in advance of the regenerating sprouts. Furthermore, it has been shown that "super-regeneration" can be readily induced in such an irradiated blastema by suitable injury, the rate of renewed capillary growth equalling the rate of growth which initially existed for the particular blastema before irradiation. The present paper seeks to correlate morphologic changes with the quantitative results, and describes and illustrates the behavior of regenerating blood vessels submitted to subthreshold and threshold doses of irradiation, together with the changes observed in the development of fibrous barriers and during "super-regeneration." The descriptions, together with illustrations, are based entirely on the appearances *in vivo* of individual blastemata, before and after irradiation. A review of the literature dealing with the effects of irradiation on vascular regeneration, has failed to yield significant direct evidence of this nature, based on continuous microscopic observation of living regenerating vessels. Histologic studies of fixed tissues fail to provide a dynamic picture of the events which take place, both quantitatively and qualitatively, and do not offer satisfactory information in assessing the tactical behavior of individual cells and cell groups during regeneration in normal or abnormal circumstances.

MATERIALS AND METHODS

The ear chamber used in these experiments has been described elsewhere.[15] The entire chamber is a modified Sandison-Clark chamber. The component parts are constructed from perspex, including cover, pillars, and retaining nuts. This enables uniform irradiation of the contained tissues

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without "hot spots" due to scatter from any metal components. The chamber and surrounding tissues were irradiated with either 200 kv. roentgen rays or gamma rays from a Co^{60} source, the latter being applied as a "sandwich mold." The dose received by the tissues can be accurately calculated with backscatter, and the variation in tissue dose was less than 1 per cent. Actual dosage estimations in tissue were checked with direct measurements made in a perspex phantom with an ionization chamber.

The insertion of the chamber at operation, follows the normal aseptic routine for this technique.

The animals used in these experiments were adult semi lop-eared rabbits, weighing from 2 to 4 kg., and bred from a fawn-colored strain of lop-eared animals originally obtained from the M.R.C. Laboratories, Mill Hill. The rabbits were housed in separate cages on grids in a temperature controlled animal house at 70°(±2°) F., and fed on a pellet diet (Bruce and Parkes No. 2) with added greens and liberal water supplies.

Usually each animal had one chamber inserted in each ear. The central chamber table (6 mm. diameter) was positioned at operation to be situated adjacent to the central artery at approximately the level of middle and distal thirds of the ear. The observations on growth were confined to the tissues growing into the space (50–100 μ in thickness) overlaying this central table.

Irradiation of Ear Chambers. The irradiation was given as a single dose with the animal unanesthetized and comfortably held in the normal sitting posture in a rabbit holder. The tissue dose administered varied from 200 r to 5,000 r. The irradiation was usually given seven to ten days after the blastema had made its appearance onto the table. This initial period was used to study normal appearances, measure growth rate prior to irradiation and ensure satisfactory control conditions. Further observations were made for periods up to eighteen months after irradiation. Irradiation of the blastema after a partial period of growth has certain advantages, apart from comparisons of growth rate and microscopic appearances before and after irradiation. If the chamber is irradiated immediately after operation or during the latent period before growth appears on the central table, a "failed" chamber, due to infection or errors in operative technique, may be misinterpreted as due to radiation damage. Furthermore, such irradiation would almost certainly add to the incidence of infection, which may occur with this technique. Irradiation of the intact ear prior to the operation of insertion of the chamber is beset with similar difficulties.

Experimental Techniques. Essentially, results were based on long continued microscopic observation of individual capillary sprouts and related vessels using both low and high power magnifications, together with photomicrography of the same field at times dictated by events. The animal was never anesthetized during these procedures. Certain addition techniques were adopted:

1. Photography of the whole table area at daily or two day intervals throughout the period of growth; this enabled a comparison of the general vascular pattern before and after irradiation, quantitative assessment of growth rates,[14] and provided a more general appraisal of the changes in vascular pattern and form which took place during the repair process.

2. High speed electronic flash photomicrography "stopped" movement of the blood and pulsation of vessels, and gave a clarity of photographic detail unequalled, in the writer's experience, by other modifications of the optical technique.

3. The use of polarized light microscopy enabled identification of certain birefringent structures of sufficient retardation (particularly collagen fibers). The writer is indebted to Dr. A. G. Sanders, Sir William Dunn School of Pathology, Oxford, for this valuable suggestion. Attempts to measure the birefringence of maturing collagen *in vivo* using an elliptical compensator of the Köhler type proved to be of limited value only, owing to difficulties experienced in

orientating the animal so as to align the fiber in the correct azimuth of polarization, and the even greater difficulty in accurately assessing fiber thickness for the purposes of quantitative estimates.

4. Vital staining of macrophage-histiocyte systems using vital new red, or Evans blue according to the technique of Ebert and Florey,[5] facilitated observations of the recruitment and maturation of individual cells of this system. The use of a chamber base in which previous irradiation and aging had caused a mosaic pattern of surface cracks to appear on the table face proved to be invaluable for the rapid identification of the same portion of the repair blastema, and the same cells, from day to day. A detailed account of the effect of ionizing radiations on macrophage-histiocyte systems and recruitment from monocytes is not included in this paper but will be published later.

Wounding and Manipulation of the Irradiated Repair Blastema. Attempts made to induce focal mechanical trauma to a portion of the irradiated tissues present in the chamber space by removal of the cover under buffered solutions so as to provide suitable exposure of the tissues proved unsatisfactory. Extensive vascular stasis and tissue damage usually resulted, and most chambers became infected. The use of a chamber with an access hole and removable pin, in a fixed position, was unsuitable in dealing with a selected focus in the tissues.

The most satisfactory results were obtained by simple firm digital compression of the chamber surfaces. This manipulation causes areas of tissue destruction and hemorrhage, ranging from small petechiae to more extensive areas of tissue damage. Fortunately, in 3 ear chambers, a focal point of damage was caused to a selected site by localizing the point of compression to the cover opposite the desired tissue area (*vide infra*), but this experiment could not be duplicated readily for other chambers, more extensive damage resulting from the manipulation.

Vascular Remodelling. Detailed studies of individual capillary fields were made by Clark and Clark[3] using a laborious camera lucida technique. Such observations have not been made, but serial photography of selected portions of the blastema provided suitable information. However, the most useful information was provided by serial photography at 8–20 diameters magnification of the *whole* table area. By this means, changes in the pattern of the larger vessels (arterioles and venules) with time, *i.e.*, remodelling, can be assessed; it is doubtful if the information provided by a detailed analysis of the changes in pattern of individual capillaries and small blood vessels provides more suitable information.

Limitations to Interpretation of Results. The total size of the repair blastema on which observations were based was governed by the size of the chamber space (*viz.* 6 mm. in diameter, 50–100 μ in thickness). Under the conditions of the experiment, the normal time taken for unirradiated chambers to regenerate was three to six weeks, depending on eccentricity of growth, positioning of chamber and individual variation in rate of regeneration among animals. It follows that for subthreshold doses of irradiation which do not precipitously inhibit growth, postirradiational observation of the growing phase is limited to a period somewhat less than three to six weeks depending on the time selected for irradiation. The interpretation of the results which have been obtained, therefore, must be strictly related to repair of areas of tissue damage of comparable size (*vide infra*) within a circle of irradiated tissue corresponding to the size of field irradiated (3.1 cm. in diameter circle in the case of the Co^{60} source, 3.0 cm. in diameter circle for roentgen irradiation).

Also, the irradiation is given during the progressive growth phase of the repair blastema. Whether corresponding effects, particularly quantitative effects, would result from irradiation applied during the latent and demolition periods after wounding, or when the tissues are still intact before wounding, cannot be stated. However,

the results of induction of trauma in the static irradiated blastema, whether growth is partial or complete, and the phenomenon of super-regeneration gives cogent information in this regard.

The results which follow are based on observations made in 84 irradiated ear chambers. Normal vascular regeneration in unirradiated tissues was observed in a series of ten control animals and also in the irradiated animals for the period of growth preceding irradiation. The morphologic changes attributable to irradiation presume accurate evaluation of normal appearances in ear chambers, and despite an extensive literature (reviewed by Williams[17]) dealing with capillary sprouting and regeneration, confusion and conflicting opinions still attend essential aspects. Therefore, attention will be drawn to certain features of vascular regeneration observed in normal chambers by the writer, which have an intimate bearing on the results obtained.

RESULTS

1. VASCULAR REGENERATION (UNIRRADIATED BLASTEMA)

At the free "edge" of the blastema, capillary sprouts grow out from the terminal vascular arcade into the zone of discontinuity. The sprout initially consists of a solid cytoplasmic thickening, at this stage usually devoid of nuclei. As it enlarges, it elongates, and a change in optical density of the outer and central zones defines a peripheral *ectoplasm* and a central *endoplasm*. At this stage, endothelial nuclei from the parent vessel have usually *migrated* into the peripheral ectoplasm. The formation of the vascular sprout and its prevailing *form* is modified by the surrounding tissues and substances. When the sprout develops among the fibrin network in a blood coagulum (*e.g.*, at the edge of the blastema growing into a zone of free hemorrhage or blood clot), it takes on the exhuberant form depicted above, and fibrin threads form an anchoring scaffold for migration of the sprout cytoplasm in a manner analogous to migrating fibroblasts in a similar situation. Furthermore, under such circumstances, the endoplasm of the sprout is freely permeable to blood corpuscles originating from the parent vessels. The corpuscles are to be seen within the sprout in a state of stasis, often in rouleaux formation, and gradually but persistently, traverse the length of the sprout into the zone of discontinuity to form a *free hemorrhagic zone* at the edge of the blastema. This zone is seen in every normal regenerating ear chamber (Fig. 2*a*, 3*a*, 10*a* and 12*a*).

It is an intrinsic manifestation of the growth of capillary sprouts into a coagulum consisting of blood plasma clot, and has been described by most workers dealing with ear chamber techniques.[14,15,17] The construction of the chamber used in these experiments, possesses great inherent rigidity and gives rise to minimal degrees of drift in the ear, and fails to support the view that the advance hemorrhagic zone is an artefact due to tissue instability and continuous trauma. The hemorrhagic behavior of vascular development in ontogenesis, in many ways, parallels the regeneration findings.

On the other hand, if the capillary sprout forms in a zone of tissue which consists of mature avascular fibrous tissue resulting from vascular thrombosis due to various causes, morphologic appearances are very different. The sprout is blunt, and the growth less exuberant in appearance. The sprout is smaller than those to be seen at the zone of discontinuity, a clear distinction of endoplasm and ectoplasm is still possible, but corpuscular permeation is limited to an occasional diapedesis, the free hemorrhagic zone being absent. The sprout either imperceptibly "bulges" as a blind extension of the vascular lumen, or more usually takes the form of increasing length of a capillary loop—named "looping."[4] Occasionally, an intermediate appearance is seen. The sprout consists of an endothelial thickening, containing several nuclei which have migrated from the endothelium of the stem vessel, but corpuscular penetration is absent. In

this way a new vascular loop gradually forms. The latter mechanism is the one almost always adopted in vascular remodelling, and also plays a dominant role in the organization of "fibrous barriers," which surround areas of tissue damage due to chronic infections, *e.g.*, tubercles, and certain other damaging stimuli (*vide infra*).

The exuberant sprout which originates at recent zones of discontinuity maturates by gradual resorption of the central endoplasm, and with further migration of nuclei into its ectoplasm, forms a hollow nucleated tube in continuity with parent vessels. The sprouts join and link up with neighboring sprouts and vessels and a circulation is established. Individual sprouts may give rise to secondary sprouts.

The type of sprout formation which organizes zones of tissue discontinuity with hemorrhagic advance is accompanied by intense macrophage formation and activity, the latter being largely concerned with the absorption and phagocytosis of this zone of "new" hemorrhage, precipitated by vascular regeneration. The linear growth rate for this type of sprout formation[14] is much faster than growth rates observed for "looping" and related types of nonhemorrhagic sprout formation (Fig. 11 and 12, *a–f*) and, indeed, pathologic circumstances which fail to permit the establishment of hemorrhagic sprout regeneration often lead to cessation of regeneration and healing. The latter has been observed in unirradiated ear chambers which recover from pyogenic infections, and also as a sequel to irradiation (*vide infra*).

Prolonged and repeated observations of endothelial nuclei, both in sprouts and in vascular arcades giving rise to sprouts, have failed to identify, beyond doubt, mitotic division of an endothelial nucleus and to establish whether cell division plays a significant role in the tactical aspect of endothelial regeneration. That protoplasmic synthesis is an active factor in endothelial regeneration is supported by the large size of the terminal vascular arcades which give rise to sprouts, and by the progressive increase in size of the sprouts, whether the latter contain nuclei or are simple anuclear protoplasmic extensions. The free mobility and migration of endothelial nuclei in the ectoplasm of sprouts and in remodelling vascular endothelium has been confirmed by continuous observation of individual sprouts and capillaries. Nuclei have been observed to gradually enter the sprouts, bypass one another or even retract back from a sprout or regressing vessel into parent endothelium. Also, an apparent adhesion of sprout cytoplasm to fibrin threads is seen under favorable circumstances in thin chambers, and gives a fan shaped or attenuated contour to the sprout termination. The similarity between such behavior and migrating fibroblasts both *in vivo* (Fig. 4) and *in vitro* is striking, and supports the view held that migration of vascular endothelium is of fundamental importance in the cellular tactics in regenerative growth of blood vessels.[15] It is apparent that the effect of any agent on regenerative growth from a dynamic viewpoint must be related to those morphologic changes and tactics which cells undergo in tissue restitution and regeneration. It follows that the observations on endothelial regeneration, recorded above; namely, the respective roles of cell division, cell enlargement and cell migration, must be taken into account in assessing the effects of radiation on vascular regeneration. A further aspect of equal importance resides in the effect of different cellular environments of vascular regeneration—particularly in relation to hemorrhagic sprout formations and organized vascular or ischemic tissue barriers.

II. IRRADIATED CAPILLARY SPROUTS

A single dose of roentgen or gamma rays of less than 400 r failed to elicit observable change in growing capillary sprouts. Doses ranging from 400 to 2,000 r caused considerable immediate damage to sprouts particularly in the 1,000–2,000 r range.

The damage caused took various forms. In some cases the sprout became rounded and retracted into parent vessels over a

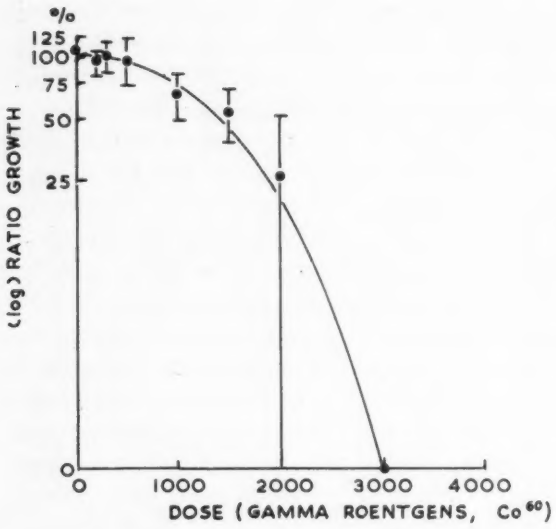

FIG. 1. Dose-response curve for repair blastemata in the rabbit, irradiated with gamma rays from Co^{60} source. The means and standard deviations are plotted, each point representing the mean of the linear growth rate ratios for at least six ear chambers.

period of several hours or days, no further growth of the sprout occurring. Alternatively, stasis of corpuscles in the sprout endoplasm and often in the adjacent capillary loops took place, with hemolysis. Subsequent changes sometimes resulted in recovery of the sprout with re-establishment of the circulation in the loops and further sprouting. Usually, however, despite re-establishment of the circulation in parent arcades, the sprout failed to progress and became isolated (pinched off) as inspissated material in the hemorrhagic zone to undergo organization by macrophages, and become obscured by compensatory sprouting with hemorrhagic advance from adjacent arcades.

Such compensation for the damage which had occurred was rapid, and was brought about either by local reconstitution of sprouts apparently partially but reversibly damaged, or by growth of sprouts showing no apparent damage, or by the formation of new sprouts after irradiation. The efficiency with which the blastema compensates for focal damage, usually resulted in an unretarded measurable growth rate after irradiation, from a quantitative standpoint[14] up to a threshold dose of 2,000 r, beyond which the growth rate of the blastema rapidly fell to zero, the dose effect curve being of an "all-or-none" (threshold) type (Fig. 1). While the value of this threshold dose, which completely inhibits blastemal regeneration, showed considerable individual variation for different animals and ear chambers, it was found that for subthreshold doses, the rate of regeneration in the chamber was unimpaired after irradiation, while doses which exceeded the threshold caused complete cessation of growth with varying degrees of destruction of the blastema already formed prior to irradiation. The sharp quantitative difference in measurable growth which took place after subthreshold and threshold dosage was reflected by the degree of damage suffered by the capillary sprouts and loops.

Effect of Subthreshold Doses. Immediately after irradiation many terminal loops and projecting sprouts appeared dilated, with stasis of the corpuscular contents, resulting in a hemolyzed fluid residue filling the loops. There was no obvious endothelial injury demonstrable in such loops, nor was leukocyte or platelet sticking prominent immediately after irradiation. With lower doses, the stasis in terminal loops recovered and the circulation recommenced, hemolyzed material being washed through by the blood. The further behavior of these vessels was consistent with normal, active growth, with hemorrhagic permeation taking place in the usual way. Recovery was usually complete one hour after irradiation. However, with higher doses, similar changes occurred spontaneously and at random along the regenerating front with similar recovery.

Alternatively, some loops and sprouts in which stasis had appeared failed to recover; a gradual absorption of the contents took place, accompanied by a retraction of the endothelium of loop or sprout, back into parent vessels. Sometimes re-establishment of the circulation in a loop was followed by retraction; new sprout and loop

formation in adjacent capillaries fully compensated for such loss. Many of the affected sprouts or loops were observed to "break off" and lose continuity with parent vessels to form inspissated debris, which were subsequently phagocytosed by clumps of macrophages. However, retraction with conservation of endothelial protoplasm was by far the commonest finding.

With higher subthreshold doses altered sprout formation appeared, similar to that seen after threshold doses, but compensatory sprouting so adequately offset the damage that quantitatively, the measured growth rate was largely unaffected after irradiation.

Threshold Doses. While changes in capillary loops and sprouts occurred and were similar to those described above, more profound changes in the whole sprouting vascular front resulted in an inadequate compensation for the damage suffered. The early changes following irradiation are illustrated in Figure 2, *a–d*. Although attempted sprout formation continued, migration and synthesis of sprout substance was markedly retarded. The sprout became either short or blunt in appearance, or the normally elongated sprout with the usual fan shaped termination became rounded or pointed. The peripheral ectoplasm appeared more clearly defined, and nuclear migration into the sprout was often impaired. The adherence of sprout endothelium to the fibrin network, which normally gives the ill defined fan-shaped appearance to the terminal portion of the sprout, was largely inhibited, resulting in rounded well defined ends (Fig. 2*c*).

Attempts at formation of secondary sprouts continued during the days following irradiation, but were incomplete or abortive with subsequent retraction of the sprouts (Fig. 2*d*). An individual sprout showed little alteration in form, or remodelling, and eventually retraction into parent vessels usually took place.

The most striking feature presented by the irradiated sprout consisted of a gross inhibition of corpuscular permeation commencing from the time of irradiation. This resulted in an absence of progressive addition of corpuscles to the advance, hemorrhagic zone. The zone, already formed prior to irradiation, was subject to continued removal by accumulated macrophages. Consequently, the spaces between sprouts became clearer, and the sprouts appeared much more prominent (Fig. 2, *b–d*). Meanwhile, fibroblasts, newly recruited macrophages and wandering cells continued to migrate into this zone, now cleared of hemorrhage and fibrin, and a "*fibrous barrier*" was initiated (*vide infra*). The early changes are illustrated in Figure 2 and the later events in Figure 3.

Once the fibrous barrier developed, normal sprout formation with advancing hemorrhage ceased, and any subsequent vascular migration and growth took the form of modified sprout formation and looping, already described above, in connection with vascularization of ischemic tissue zones.

III. "FIBROUS BARRIER" FORMATION

Following on the early changes, resulting from threshold doses of irradiation and described above, *i.e.*, alteration in sprout permeability to corpuscles and phagocytic removal of existing hemorrhage and cellular debris, fibroblast migration continued into this avascular zone in a manner similar to that taking place with normal regeneration (Fig. 4). The accumulated fibroblasts, together with a matrix composed of degenerated cell remnants admixed with secretory products, formed a dense layer which acted as a barrier to further effective sprout formation. Within a period of twenty-four hours, after the disappearance of the hemorrhagic zone around the sprouts, examination of this barrier under polarized light revealed a deposition of birefringent fibers, morphologically indistinguishable from mature collagen (Fig. 5). Individual fibers were positively birefringent and gave extinction angles, using an elliptical compensator of the Köhler design, comparable to those given by unirradiated collagen

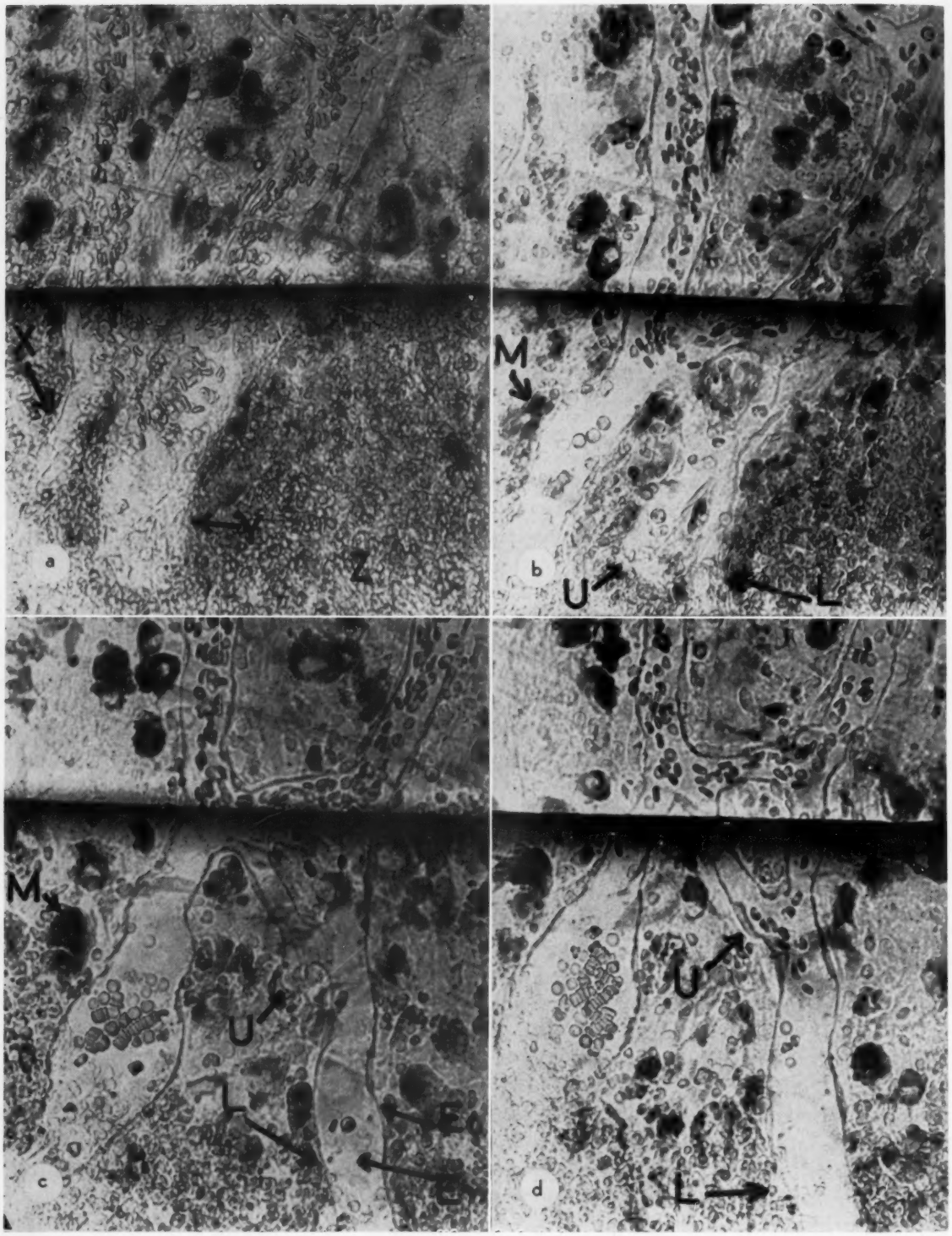

FIG. 2. Early changes in capillary sprouts after irradiation with a "threshold" dose of 3,000 r (gamma rays, Co^{60} source). The four photographs have been taken of the same group of sprouts. Several intravenous doses of Vital New Red were given to the animal to stain macrophages. The black line crossing each photograph is a marker on perspex table to aid identification of a given field. (Electronic flash photographs ×400.) (*a*) Twenty-one hours after irradiation. Two newly formed sprouts (X, Y) in lower portion of photograph embedded in hemorrhagic zone (Z) and growing downwards. The sprouts are blunt and more well defined and rounded than is usual in unirradiated chambers, and are relatively empty. (*b*) Forty-four hours after irradiation. Practically no effective growth although sprout Y has bifurcated (U, L) and sprout X is

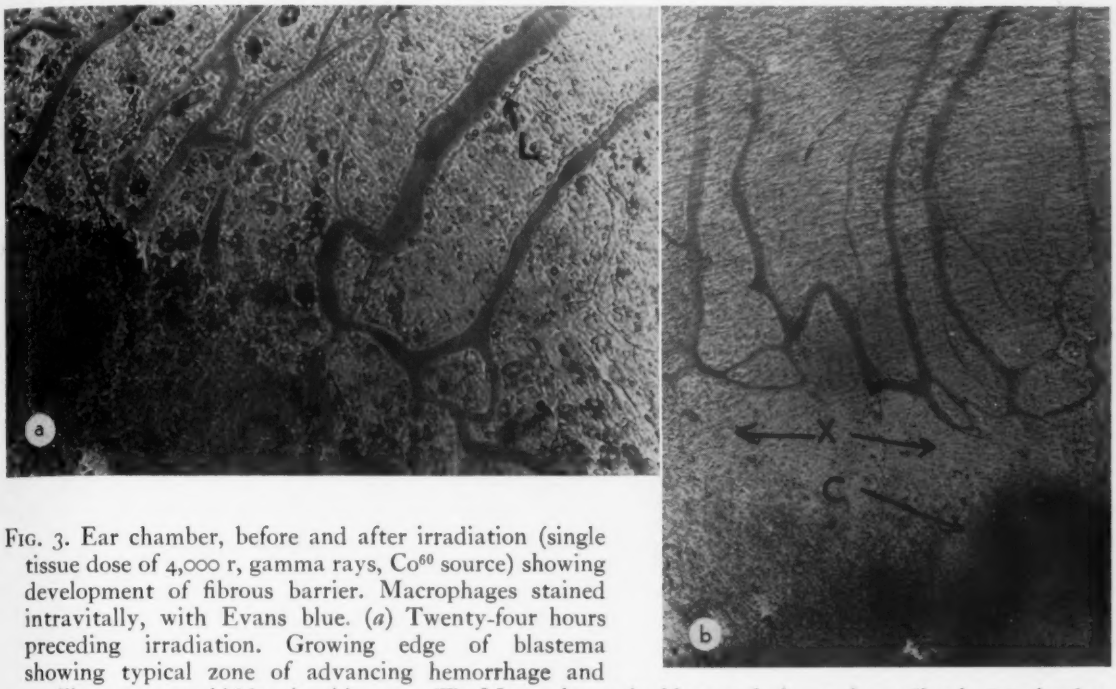

Fig. 3. Ear chamber, before and after irradiation (single tissue dose of 4,000 r, gamma rays, Co^{60} source) showing development of fibrous barrier. Macrophages stained intravitally, with Evans blue. (*a*) Twenty-four hours preceding irradiation. Growing edge of blastema showing typical zone of advancing hemorrhage and capillary sprouts hidden in this zone (Z). Macrophages in blastemal tissues immediately proximal to hemorrhagic zone proceeding with demolition of hemorrhage. Emigrating leukocytes and monocytes (L) stuck to vascular endothelium. (×170.) (*b*) Larger section of same vascular front at lower magnification (×80), 360 hours after irradiation. Hemorrhagic zone has disappeared due to inhibition of new sprout formation and demolition, and a dense fibrous barrier (X) has formed in advance of growing vascular front. Further effective capillary sprouting has ceased. Old clot at C heavily colored due to intravital staining.

fibers in ear chambers. Strict quantitative assessment of the light retardation in these fibers was not satisfactory, and only a very approximate estimation of the birefringence could be made. However, polarized light examination of the advance zone in normal regenerates has failed to reveal similar birefringent fibers, the formation of orientated collagen always lagging appreciably behind the vascular sprouts in the regenerating blastema in agreement with previous observations.[10,11] The pro-collagen matrix which closely surrounded the contained vascular buds was characteristically isotropic (Fig. 5*b*).

The fibers in the barrier zone were orientated in bundles at right angles, similar to the arrangement in the mature blastema, most bundles however running parallel to the terminal vascular arcade (Fig. 5*b*).

slightly larger. Marked macrophage activity is resulting in demolition and progressive clearing of hemorrhagic zone, which causes the sprouts to appear better defined. Some leukocytes have stuck to walls of vessels and sprouts, and further recruitment of macrophages (M) from monocytes has occurred. (*c*) Sixty-eight hours after irradiation. Further demolition and clearing of hemorrhagic zone. The sprouts are now well pronounced in cleared background; peripheral ectoplasm (Ec) contains endothelial nuclei, and central endoplasm (En) contains a few clumps of erythrocytes in rouleaux formation together with leukocytes. Note (1) the relative absence of growth of sprouts, and (2) the alteration in form of bifurcated sprout Y, the sprout L having extended at the expense of sprout U ("remodelling"). (*d*) Ninety-two hours after irradiation. Further demolition of hemorrhagic zone despite cessation in growth of sprouts with early formation of what eventually becomes an avascular fibrous zone in advance of the sprouting front. Endothelial redistribution has caused the formation of a single elongated sprout L, with disappearance of the sprout U. Comparisons of the sprouts in *a* and *d* clearly show that no effective growth has taken place despite remodelling of the sprout system as a result of endothelial migration. However, hemorrhagic permeation of the sprouts has been inhibited despite active flow of blood and little change in caliber of the terminal vascular arcades. Demolition of the formed hemorrhagic zone has proceeded largely as the result of macrophage activity. Note stasis in sprout at left by comparing the rouleaux formations with *c*.

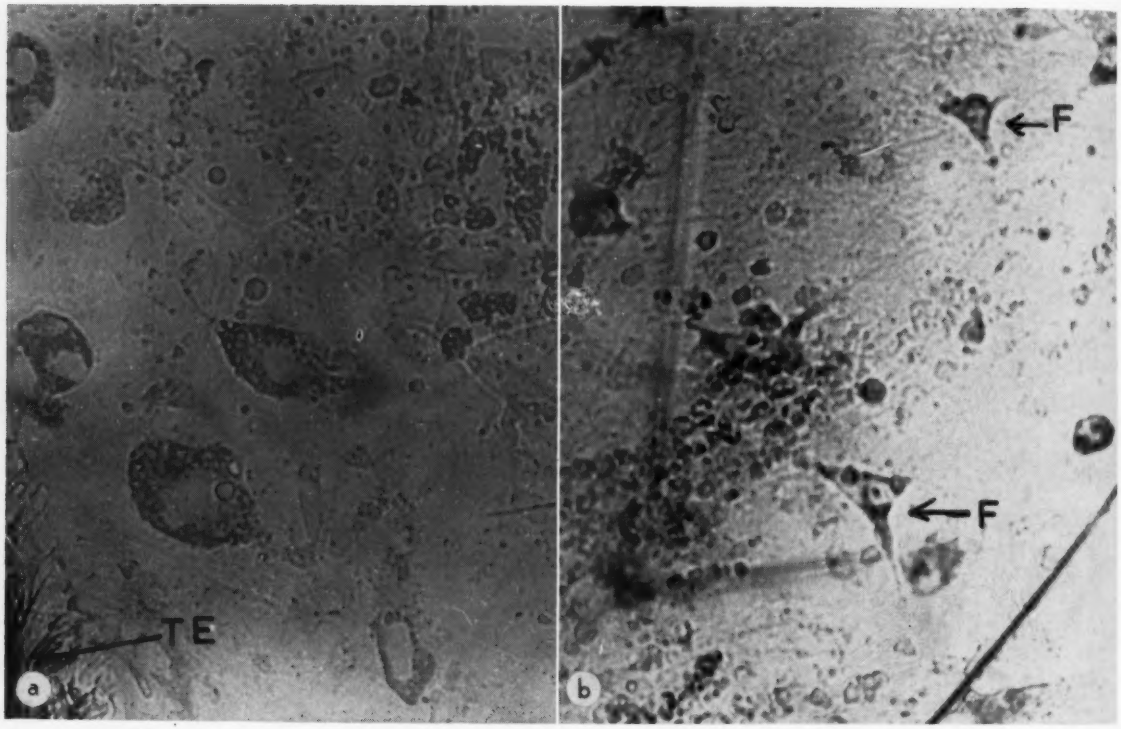

FIG. 4. Migrating cells in sprouting zone in an irradiated chamber—48 hours after 2,000 r, during demolition period. (Electronic flash photograph ×900.) (*a*) Newly formed macrophages of Lewis and Lewis type, containing two types of granules and erythrocytes (lower cell). Cells not stained intravitally. Table edge (TE). (*b*) Migrating fibroblasts (F) among cellular debris.

Often the fibers were coarser than those seen in the unirradiated blastema.

The capillary sprouts and loops embedded in the fibrous barrier showed an altered appearance (Fig. 6, *a–d*).

Short deformed sprouts predominated (Fig. 6*a*) and were placed irregularly along the vascular front (Fig. 3*b* and 6, *a–c*). Large sections of the vascular arcade failed to generate effective sprout formations (Fig. 3*b* and 8*a*), while at certain points, intense focal sprout development resulted in grossly aberrant appearances (Fig. 5*a* and 8*a*). Often sprouts consisted of blind tubes congested with red cells (Fig. 6*d*). Sometimes a short blunt sprout was seen containing aggregates of endothelial nuclei, while vascular looping into old degenerated vessel spaces was commonly observed in a manner similar to that described by Dodson *et al.*[4] for healing tubercles in ear chambers.

IV. VASCULAR "SUPER-REGENERATION" IN IRRADIATED EAR CHAMBERS

By firm digital compression of an ear chamber irradiated with doses of 1,000 r or more, punctuate petechial hemorrhages could be readily induced, together with areas of vascular stasis (Fig. 7). It was noted that very rapid organization of such hemorrhages occurred (Fig. 7*c*) and took place by means of sprout formation.

As a result of this observation, blastemata in which irradiation had resulted in the formation of a dense fibrous barrier, which apparently prevented further effective sprout formation, were subjected to similar traumata. It was found that for such chambers in which growth had remained "static" for periods up to six months, extensive areas of hemorrhage, produced mechanically, were rapidly organized by exuberant sprout formation and advance hemorrhagic growth, and sub-

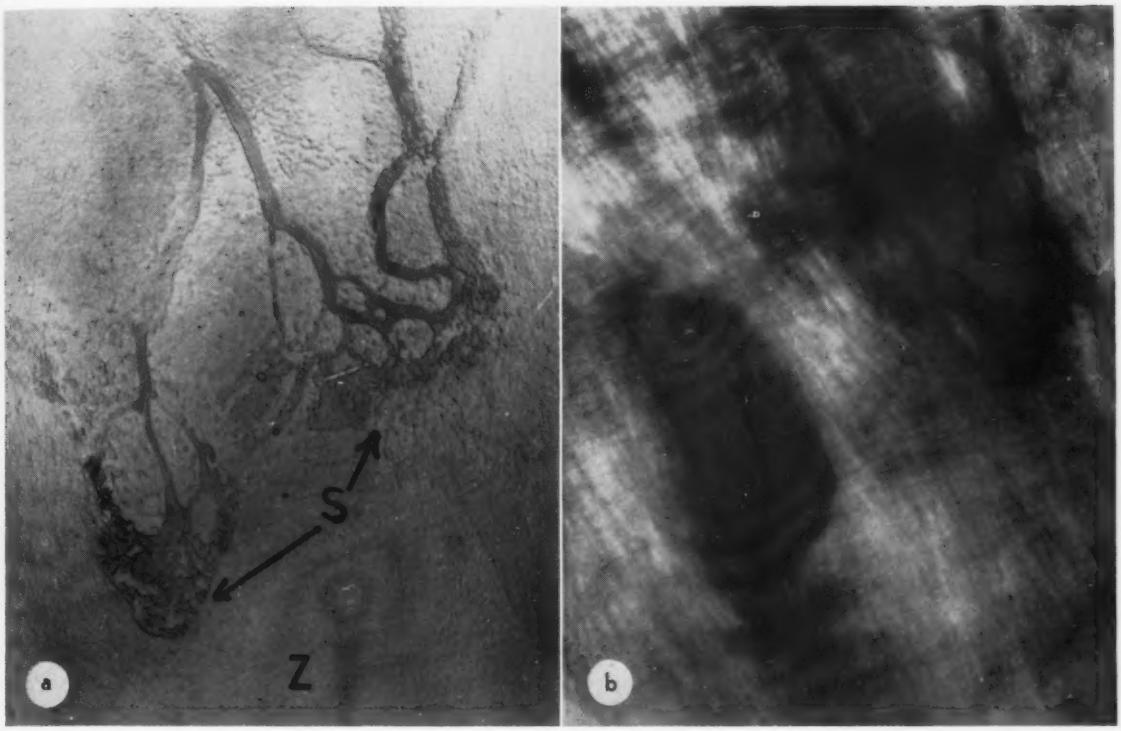

Fig. 5. Fibrous barrier zone in an ear chamber irradiated with a single tissue dose of 4,000 r (gamma rays, Co^{60} source) during regeneration. (×200.) (*a*) Photograph of terminal capillary arcade embedded in clear barrier zone (Z), taken with ordinary light. Aberrant sprout formation (S) without hemorrhagic manifestations. (*b*) Same field as in *a*. Photograph taken with polarized light. The barrier zone is seen to be permeated with birefringent fibers which appear white (or black in an azimuth of polarization of 90°), identical to the appearance of mature collagen. The zone immediately surrounding the vascular loops and sprouts is isotropic. (In an actively growing blastema, no such birefringent fibers can be demonstrated in the hemorrhagic zone surrounding the capillary sprouts and actively migrating fibroblasts. Birefringent collagen only appears after local demolition is complete and when the blastemal tissues remodel and mature.)

sequently the whole table area regenerated at a growth rate comparable to that which held initially, prior to irradiation (Fig. 8 and 9). The morphologic appearances of the sprouts paralleled those of unirradiated regenerates. Furthermore, vascular reorganization of the fibrous barriers by this new capillary sprout formation readily took place (Fig. 8*b*). In order to clarify the sequence of events in such "*super-regeneration*," an area of focal hemorrhage was elicited at a focal point of the vascular front embedded in the fibrous barrier, as already described (*vide supra*). The changes elicited are shown in Figure 10. Rapid regeneration took place, which emanated entirely from the focal point of damage and hemorrhage. The instigation of normal sprouting resulted in a rapidly increasing zone of advancing hemorrhage, and organization of the residual fibrous barrier was entirely due to this focal growth. Again, measurements of the rate of regenerative growth gave values essentially equal to the value obtained prior to irradiation.

V. Rate of Organization of Avascular Residues

In Figure 11, a growth curve is illustrated for a chamber in which a subthreshold dose of 1,500 r was given to the growing regenerate. Subsequently, before the tissues had covered the entire table, a massive area of vascular thrombosis resulted in a secondary avascular area, which was left to organize without inducing "*super-regeneration*." The rate of organization of this avascular area was greatly retarded. Photographs of the table area for this same blas-

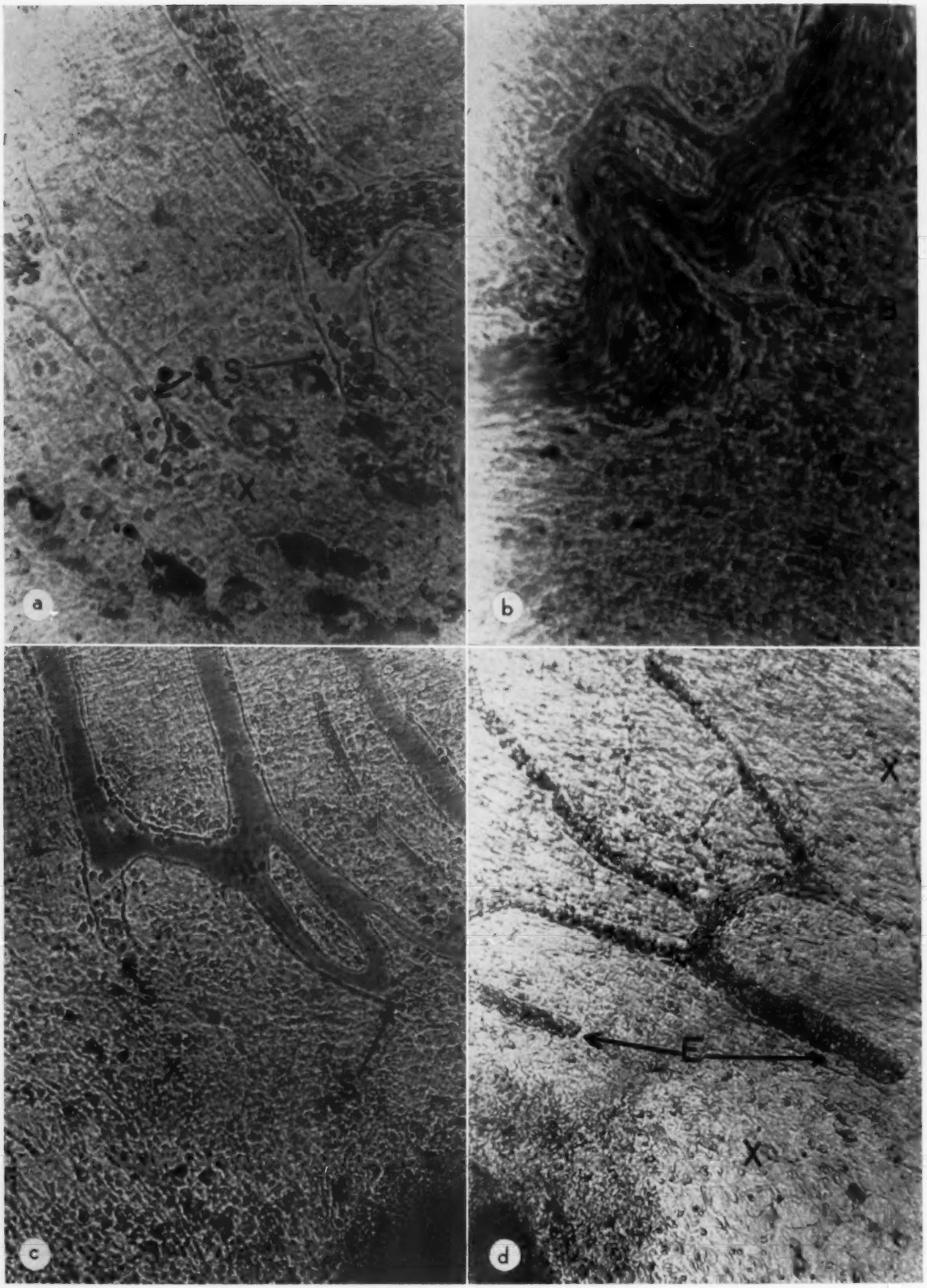

FIG. 6. Appearances of sprouting front at various stages of fibrous barrier formation in same ear chamber. (*a*) One hundred ninety two hours after 4,000 r (gamma rays, Co^{60} source). Macrophages vitally stained.

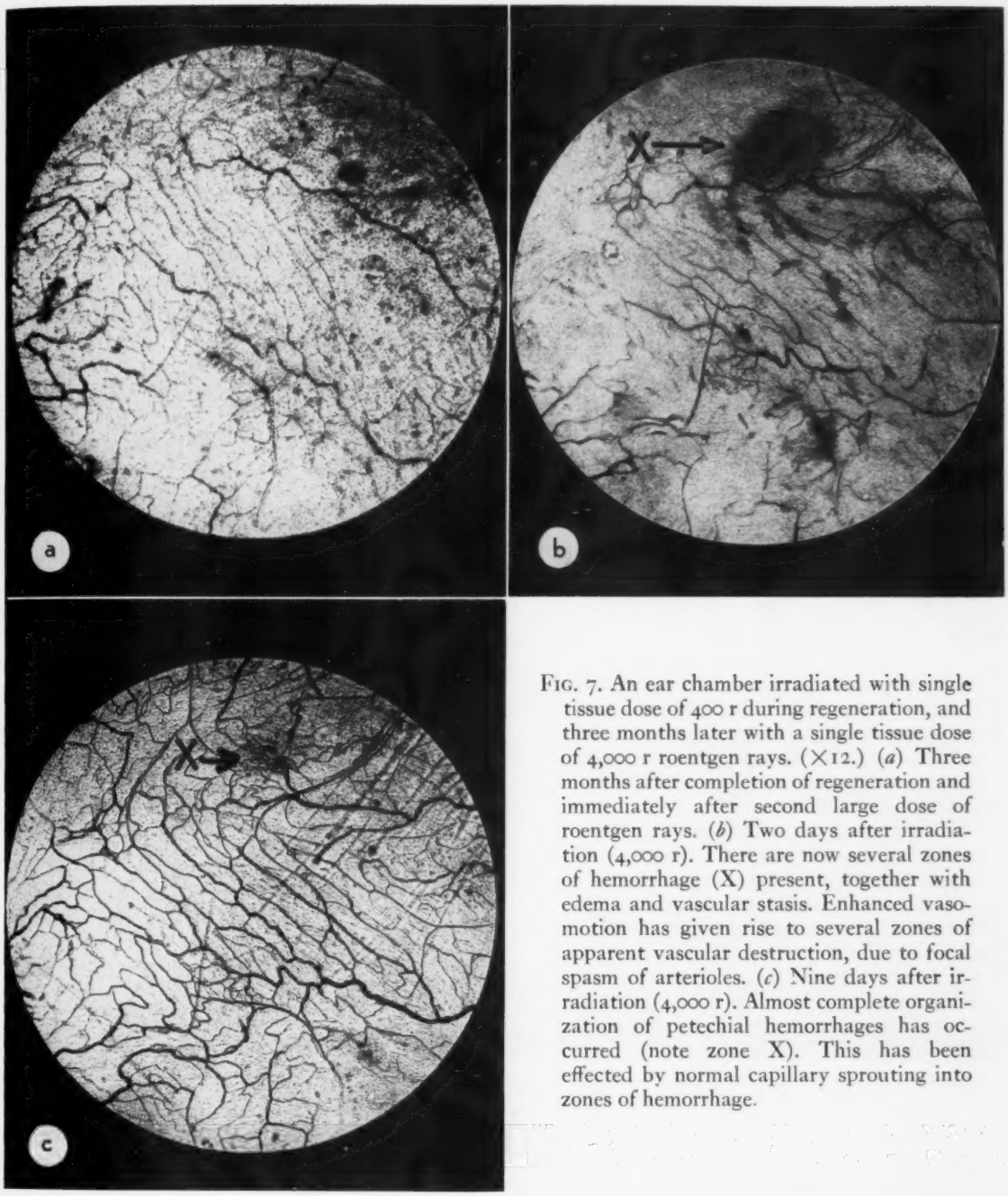

Fig. 7. An ear chamber irradiated with single tissue dose of 400 r during regeneration, and three months later with a single tissue dose of 4,000 r roentgen rays. (×12.) (*a*) Three months after completion of regeneration and immediately after second large dose of roentgen rays. (*b*) Two days after irradiation (4,000 r). There are now several zones of hemorrhage (X) present, together with edema and vascular stasis. Enhanced vasomotion has given rise to several zones of apparent vascular destruction, due to focal spasm of arterioles. (*c*) Nine days after irradiation (4,000 r). Almost complete organization of petechial hemorrhages has occurred (note zone X). This has been effected by normal capillary sprouting into zones of hemorrhage.

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Two abnormal shaped sprouts (S, S) embedded in early fibrous barrier (X). Macrophage demolition still proceeding in advance of maturing fibrous zone. (Electronic flash photograph ×400.) (*b*) One hundred and ninety-two hours after irradiation (4,000 r). Attempted formation of a new sprout (bud B) from terminal vascular arcade in young fibrous tissue barrier. (×400.) (*c*) Three hundred and sixty hours after irradiation (4,000 r). Active circulation with considerable leukocyte sticking is seen in terminal vessels. Two short sprouts (S, S) have formed, but growth is ineffectual in the presence of fibrous barrier zone (X). (×170.)(*d*) Three hundred and sixty hours after irradiation (4,000 r). Blind capillary extensions (E) and loops embedded in fibrous barrier (X). (Electronic flash photograph ×170.)

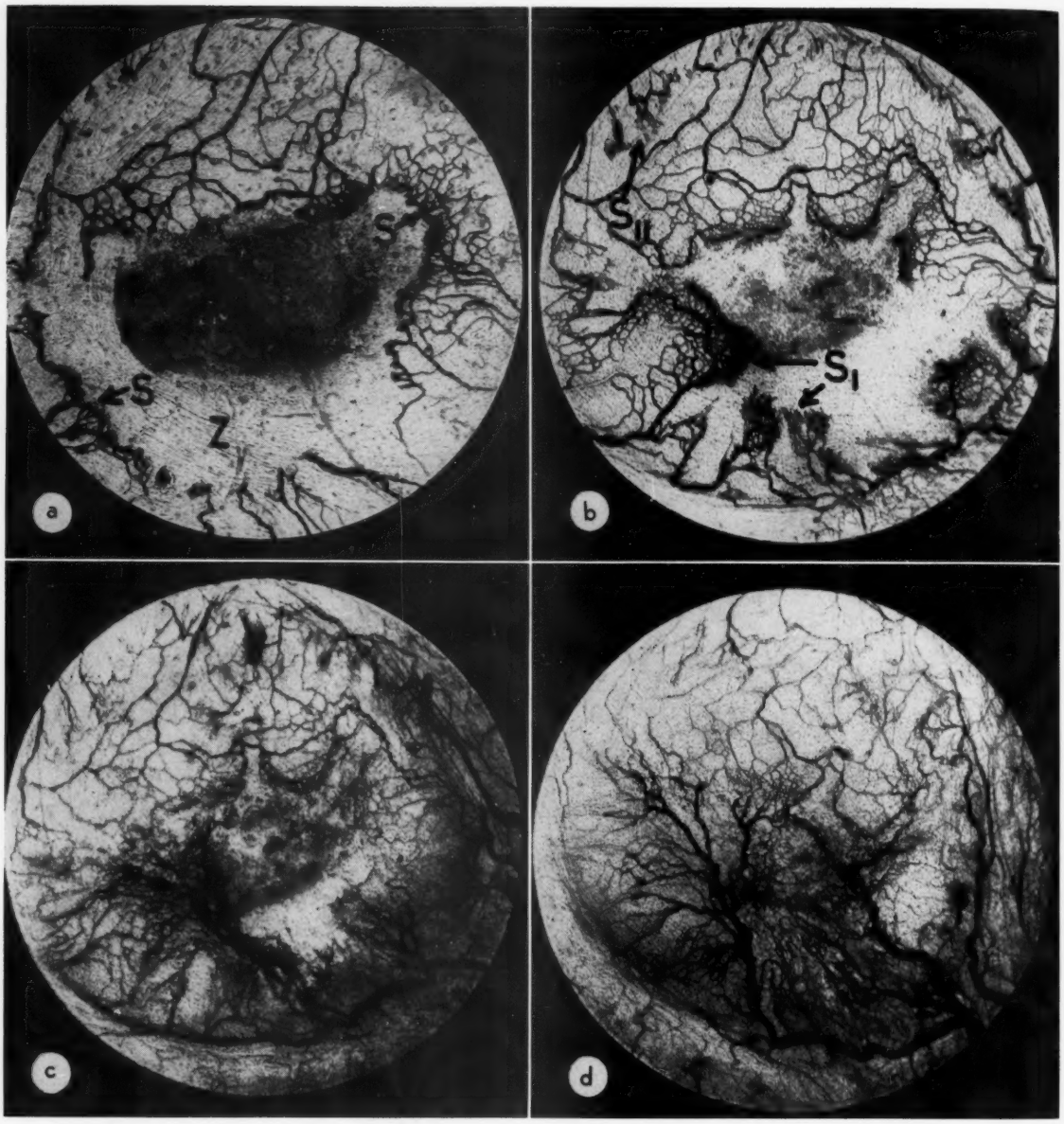


FIG. 8. An ear chamber irradiated during regeneration, resulted in formation of fibrous barrier with cessation of growth. Subsequently diffuse damage to tissue was induced by loosening chamber cover and re-tightening retaining nuts, which resulted in super-regeneration. (×14.) (*a*) Thirty days after irradiation with a single dose of 2000 r (gamma rays, Co^{60} source). Aberrant sprouts (S) embedded in thick fibrous barrier (Z). Old clot (C). No significant advance of vascular growth during preceding 30 days, despite progressive formation of avascular fibrous zone. (*b*) One hundred and twenty days after irradiation. The chamber cover was loosened and re-tightened six days previously, at which stage the appearance of chamber showed little difference from that in *a*. Note new sprout formation (S_I) with advancing hemorrhage, along whole blastemal front and similar sprouting within formed blastema (S_{II}). Rapid organization is proceeding ("super-regeneration"). (*c*) Appearance of chamber, now 138 days after irradiation. Diffuse hemorrhagic sprout formation and organization of chamber space. (*d*) Chamber, 151 days after irradiation. Complete vascularization and blastema formation. Note that interval between irradiation and *b* is 120 days, and time for super-regeneration (*b*–*d*) is 21 days. The latter period approximates to the rate of regeneration before irradiation (see text).

tema (Fig. 12) show the differences in appearance between the normal hemorrhagic sprouting zone and the nonhemorrhagic capillary growth into the avascular fibrous zone which resulted from vascular thrombosis in the formed regenerate.

Similar changes occurred in regenerates which had received threshold doses, resulting in fibrous barrier formation. The barrier inhibited normal growth as an indirect mechanism. That the intrinsic capacity for vascular growth remained intact was shown by the phenomenon of super-regeneration, induced by secondary trauma, and by the comparison of growth rates in two areas of the same regenerate (Fig. 11).

VI. CAPILLARY DAMAGE AND VASCULAR REMODELLING

Threshold and subthresold doses of irradiation in excess of about 400 r caused focal areas of capillary and venular damage. The latter consisted of an initial stasis either followed by recovery or by retraction of the vessel remnants into undamaged parent vessels. There were no signs of endothelial swelling causing primary obliteration of the lumen, as postulated by Borak.[1] The vessel degenerated *in situ*, the two surviving ends often forming blind appendages, in the tip of which platelets and leukocytes were often seen attached as thrombi to the endothelial lining (Fig. 13). The original site of the vessel could be identified as a space in the connective tissues, and sometimes a new sprout grew along this track, as described above, with reconstitution of the vessel. Following stasis in a venule, not infrequently, a leukocyte and platelet thrombus filled a major segment of the vessel. Again a gradual disappearance of such thrombosed vessels occurred, or regeneration by capillary sprout formation took place. However, after higher doses of irradiation, particularly threshold levels, recovery of damaged capillaries by regeneration was severely limited, although quite major general vascular remodelling still occurred in such a regenerate, in which the vascular index had

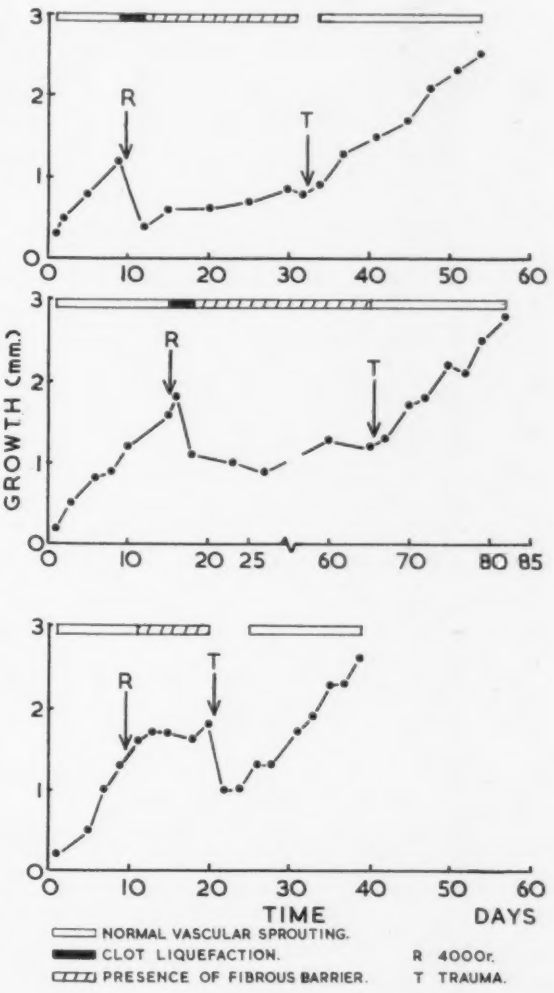
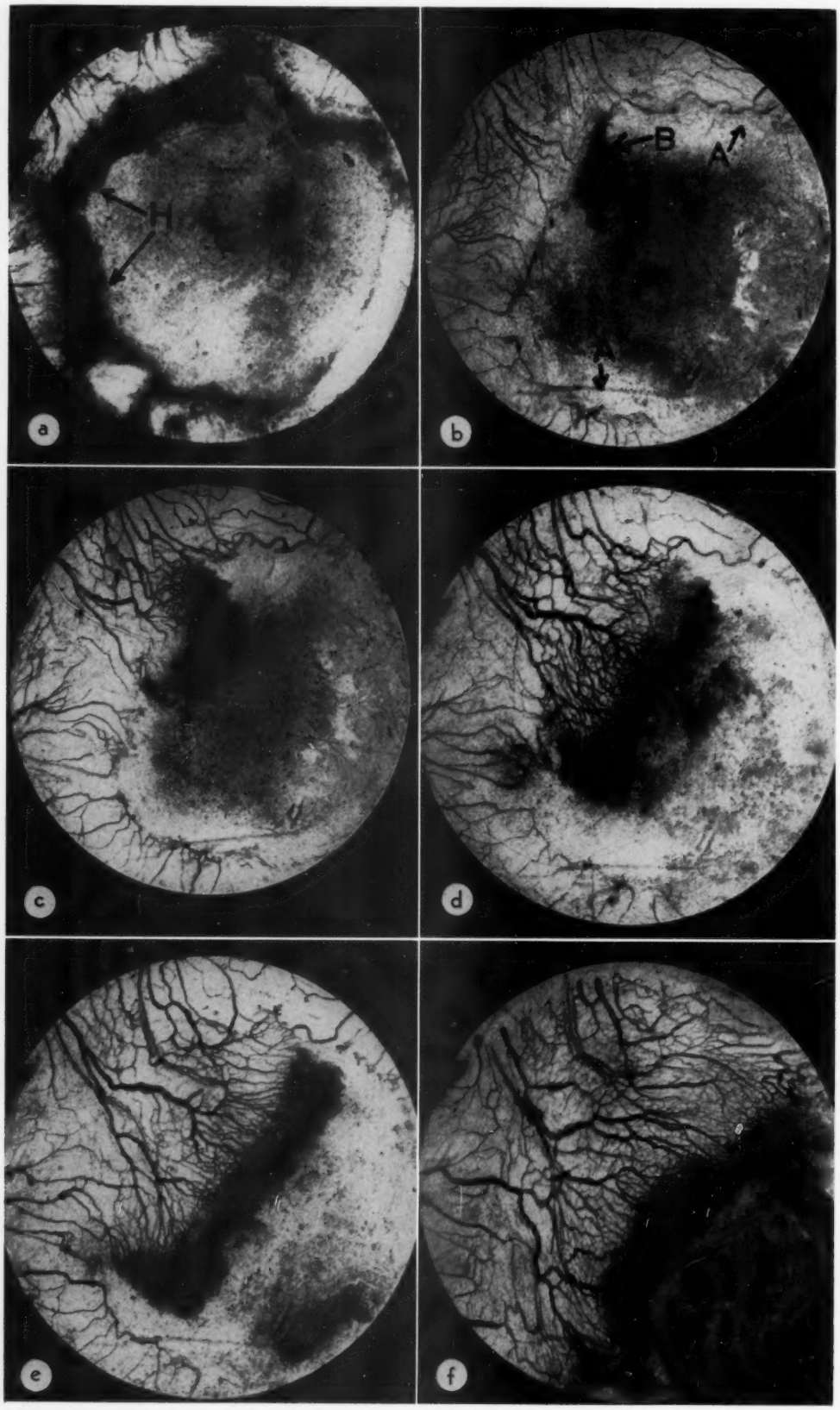


FIG. 9. Growth curves for 3 ear chambers irradiated with 4,000 r single dose of gamma rays from Co^{60} at R. In each case further vascular growth ceased after irradiation, with formation of a "fibrous barrier." At point T, super-regeneration was induced by traumatizing the surviving repair blastema by temporarily loosening the cover of chamber. In each case, regeneration recommenced and continued to completion at a rate of growth essentially equal to that prior to irradiation for the particular chamber. RT interval 22 days, 50 days and 11 days, respectively.

been considerably reduced. This was confirmed by the reconstructions in the pattern of larger vessels—particularly venules—which took place over the weeks and months following irradiation (Fig. 12, *e* and *f*). Such reconstruction (remodelling), which is considered indicative of endothelial lability in response to functional altera-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tions in accordance with the histomechanical principle of Thoma (reference, Williams[17]), is undoubtedly dependent on endothelial migration to allow for alterations in form and is probably little different from the cellular tactics governing vascular regeneration (sprouting and looping). However, remodelling was accomplished with great conservation of endothelium, and largely consisted of rearrangements of existing structures rather than synthetic additions combined with degenerations. With high doses of irradiation (3,000–4,000 r) remodelling of surviving vessels is restricted (compare Fig. 7, *a* and *c* and see also van den Brenk[16]).

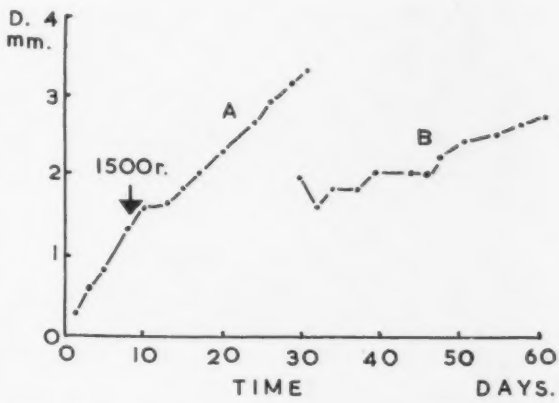

Fig. 11. Ear chamber irradiated with 1,500 r roentgen rays. Curve A indicates the mean linear growth of the repair process before and after irradiation. Curve B is the plot of the rate of repair of a spontaneous infarct, due to vascular thrombosis, which occurred after irradiation. The time base line corresponds for the two curves. Photomicrographs of this chamber are illustrated in Figure 12.

VII. Inflammatory Hyperemia in Nonirradiated and Irradiated Vessels

If an unirradiated chamber became infected, the hyperemia which resulted seemed partly due to increased corpuscular filling of the capillary *network* as a result of arteriolar dilatation, and dilatation of precapillary sphincters and metarterioles. However, the capillary caliber reached a size which seemed far beyond the elastic limits of the vessels; the latter is governed by the consideration embodied in the conception of "critical closing pressures."[2,8] There can be little doubt that the capillary caliber attained is due to hypertrophy and active growth of endothelium. The involved reconstruction of intravascular volume requires a lability of endothelial form, not consistent with simple mechanical dilatation. This was clearly seen in ear chambers.[16] The vascular pattern of the acutely inflamed tissues in a chamber is different from the dilatation induced by chemical vasodilators. The grossly enlarged vessels showed no apparent thinning of vascular endothelium, but to what extent this was accomplished by simple redistribution of endothelium or by protoplasmic synthesis (hypertrophy or hyperplasia) remains doubtful.

In irradiated chambers which became infected, the "hyperemic reconstruction" of

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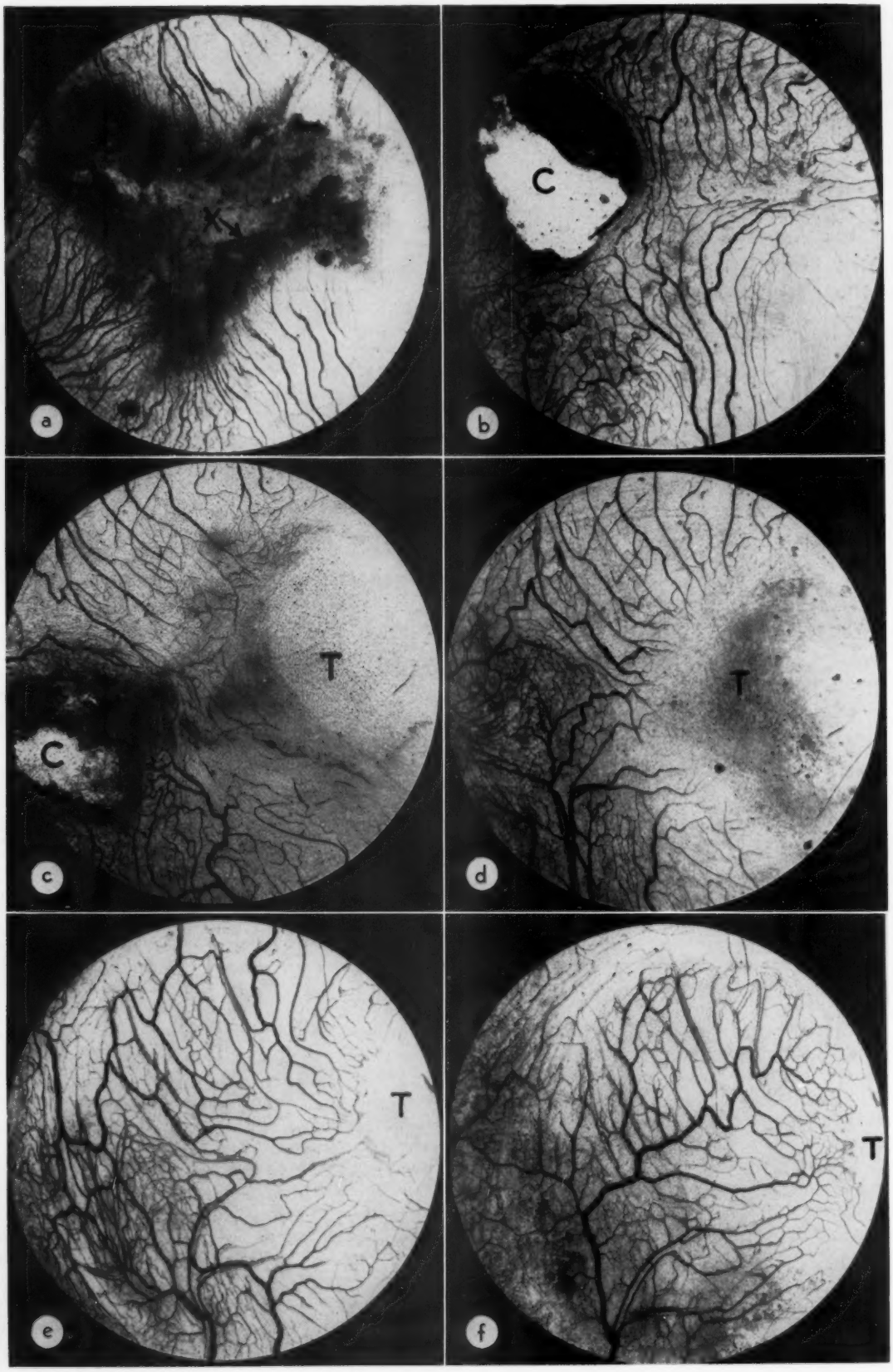
Fig. 10. An ear chamber in which irradiation with a single tissue dose of 3,000 r (gamma rays, Co^{60} source) caused cessation of growth, with the formation of a "fibrous barrier" (see text), and in which focal trauma was applied to the growing vascular front, resulting in super-regeneration. (×12.) (*a*) Five days preceding irradiation. Normal front of vascular sprouts with advancing hemorrhage (H) surrounding original unorganized clot (C). (*b*) Twenty days after irradiation. The clear zone A has formed since the irradiation and consists of fibrous tissue, which has prevented active vascular sprouting. At point B focal trauma has been applied and has induced an area of hemorrhage. Active vascular sprouting with advancing hemorrhage (super-regeneration) is confined to this zone. (*c*) Five days later (25 days after irradiation) super-regeneration has taken place from point B only and is rapidly organizing both original fibrous barrier A and unorganized clot in chamber. (*d*) Nine days later (34 days after irradiation) further stage in super-regeneration. (*e*) Five days later (39 days after irradiation), a further stage in super-regeneration. Note the organization of original fibrous barrier A from vascular growth originating from point B and also the progressive advance hemorrhage, characteristic of normal capillary sprouting. (*f*) Eleven days later (50 days after irradiation), original fibrous barrier organized by vascular growth. (This chamber proceeded to complete organization of the whole table area.)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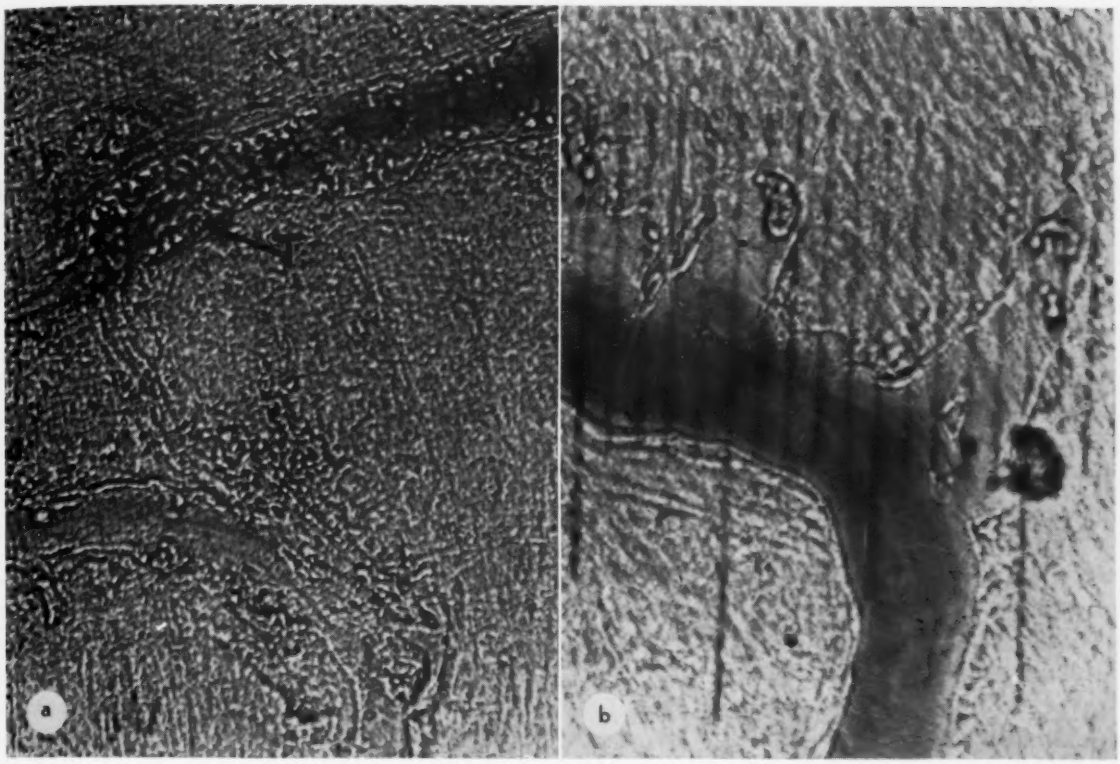

Fig. 13. Venular thrombosis and destruction following irradiation. Chamber seven days after 3,000 r (gamma rays, Co^{60} source). (×170.) (*a*) Leukocytes and platelets (T) attached to wall of venule undergoing thrombosis. (*b*) Blind vascular remnants containing leukocyte and platelet thrombi with margination of plasma and retracting into larger venule with circulation intact. (Photographic exposure three seconds. Lines in photograph are due to micrometer ruling in eye-piece.)

vessels was greatly decreased, although still present to a limited degree. Whether this was due to intrinsic endothelial damage or to indirect extravascular tissue damage is uncertain. However, the onset of acute inflammation in a chamber irradiated to higher doses usually led to massive areas of vascular damage, due to stasis, hemorrhage and thrombosis, and in chambers which had received threshold doses, necrosis of the whole regenerate was common.

VIII. VASCULAR MATURITY AND RADIOSENSITIVITY

Large doses of radiation (4,000 r) given to partially regenerated ear chambers sometimes resulted in the growth changes de-

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Fig. 12. An ear chamber irradiated with a single dose of 1,500 r roentgen rays (250 kv.). (See also Figure 11.) (×14.) (*a*) Stage of partial regeneration four hours before irradiation. Normal capillary sprouting with zone of advancing hemorrhage (X). (*b*) Ten days after irradiation. Further regeneration has occurred. Unorganized zone (C). (*c*) Twenty-one days after irradiation. Further regeneration of unorganized zone (C) but spontaneous thrombosis has resulted in an infarct at T. (*d*) Thirty-seven days after irradiation. Zone C has completely regenerated but repair of infarct at T has proceeded slowly. The sprouts are growing into an avascular fibrous residue without the normal hemorrhagic advance zone (see text). (*e*) Seventy-eight days after irradiation. Repair of infarct T is still incomplete despite an elapse of 57 days from onset of thrombosis (as shown in *c*.) (*f*) One hundred and twenty-eight days after irradiation. A small portion of the infarct (T) remains unorganized. Compare rate of normal capillary sprouting (zone X) after subthreshold irradiation (*a–c*, a period of 21 days) with slow rate of vascular regeneration, into a fibrous tissue residue (*c–f*, a period of 107 days). The respective growth curves for the two types of regeneration in this chamber are given in Figure 11.

scribed above under threshold effects. More frequently, this dose caused complete destruction and necrosis of the blastema on the central table, particularly if radiation was given to a blastema at an early stage, when only a small portion of the table area was covered by the growing tissues. When the blastema covered a larger surface area due to more advanced stages of growth, irradiation with larger doses often resulted in rapid degeneration and necrosis of the marginal sprouting zone, together with the most recently formed vascular arcades, loops, and related vessels. Subsequently, the vascular component of the surviving regenerate remodelled to form a terminal arcade, and the destroyed vascular zone was replaced, invariably, by a typical fibrous barrier zone, already described. At a later stage, the secondary radiation reaction at ten to twenty days after irradiation, typified by hyperemia, stasis, leukocyte sticking and round cell infiltration, often resulted in a delayed necrosis of the remaining regenerate. However, regenerates suviving this phase and which were subjected to secondary trauma frequently reacted by typical *super-regeneration*, with a resulting completion of growth over the whole table.

DISCUSSION AND CONCLUSIONS

In those organisms in which tissue oxygen and nutrient requirements are subserved by a vascular system, restitution, regeneration and repair of tissue damage invariably involve regeneration and remodelling of small blood vessels. Without such reconstruction and growth of the vascular system, regeneration and growth of parenchyma and stroma are strictly limited in vascularized tissues—both in orthoplastic and neoplastic states. In the case of a simple traumatic deficit of the integument, the migration and growth of epithelium cannot proceed efficiently without the formation of a vascular repair blastema (granulation tissue), and continuous remodelling of the vascular element takes place *parri passu* with epithelization, as seen in ear chamber studies *in vivo*.[15]

It follows that the primary consideration in tolerance of tissues to irradiation is the capacity retained by the irradiated vessels to regenerate and remodel. An analysis of this problem presents far reaching implications and difficulties. A simple approach would be to consider vascular endothelium as a single cellular system with an inherent radiosensitivity, which would be generally reflected by radiation induced changes throughout the body *in vivo*. That such a simple approach is clearly untenable is evidenced by the complex organization of individual cellular systems in such a relatively "simple" tissue as granulation tissue and the various direct and indirect radiation effects which demand attention, as shown in the studies presented in this paper and in a previous study.[14]

It has been shown above that the morphologic pattern of capillary regeneration varies with different circumstances. One manifestation is the exuberant, rapid hemorrhagic sprouting which invades areas of complete tissue loss or foci of pre-existing hemorrhage in tissues, as seen in a newly inserted ear chamber. Another manifestation is the slow nonhemorrhagic progression of sprouts into viable but relatively avascular zones of tissue, such as thrombotic zones and artificially induced fibrous barriers (*vide supra*). This type of regeneration, while still an example of epimorphic growth in which the intrinsic behavior of vascular endothelium simulates the hemorrhagic type, nevertheless takes on the different morphologic patterns already described as modified sprouting, bud formation and looping, and furthermore, gives rise to quantitative differences which become of greater significance when the efficiency of the mechanism is related to the survival of the tissue or organ. Yet a further manifestation is that of vascular remodelling. Here we are not so much concerned with the synthesis and growth of new endothelial protoplasm, but rather with a rearrangement of existing endo-

thelium to meet focal alterations in tissue structure and requirements. While this process may be limited in highly specialized organs in the mature organism, it plays essential roles in the survival of tissues subject to injury, in maturing granulation tissue, and in all pathologic conditions involving changes in basal hemodynamics (inflammations, secondary collateral circulations, etc.). Vascular remodelling depends on a great lability of vascular endothelium for reorientation of individual cells and groups of cells (both endothelial and vascular supporting cells). De-differentiation of cells, leading to hypertrophy, migration, and, to a lesser extent, hyperplasia, is as essential in remodelling of the vasculature as in regeneration, and endothelial cells apparently readily de-differentiate to permit the circulatory readjustments which are constantly occurring in certain tissues with environmental changes. In morphologic behavior, vascular remodelling bears a closer resemblance to morphallaxis than to epimorphosis.

In considering the effects of irradiation on vascular regeneration, each of the above manifestations of vascular growth must be taken into regard, and especially related to indirect extravascular tissue changes and their effects on the system as a whole.

It has been shown above that individual capillary sprouts subjected to irradiation behave in various ways. There is a very wide individual variation in response, both qualitatively and quantitatively. A sprout formed prior to irradiation may: (1) continue its growth; (2) cease growing for a period, and then proceed in a normal manner; (3) cease growing and subsequent changes in its cellular environment (*e.g.*, the formation of a fibrous barrier) may alter its pattern of growth; (4) cease growing and retract into parent vascular arcades, partially or completely; and (5) stasis and hemolysis of the corpuscles in the endoplasm may occur and either lead to recovery with subsequent growth, to retraction, or to degeneration *in situ* with sequestration from viable arcades and organization by macrophages. Similar events may take place in related vascular arcades, or in capillaries and venules in the formed regenerate. However, the intensity of the damage is maximal in the most immature sites (sprouts and arcades) and conforms to the postulate of embryonic sensitivity embodied in the Bergonié-Tribondeau law. The threshold for damage to individual sprouts is as low as 400 r (single tissue dose) roentgen rays, in the rabbit ear, but there is a very wide spectrum of sensitivity, not only in individual animals, but in a single blastema. Furthermore, compensation for focal damage is extremely efficient and may take place by: (1) recovery of the sprout as described; (2) focal formation and growth of a new sprout on the same portion of the vascular arcade; (3) formation of new sprouts or secondary sprouts from adjacent portions of the arcade and rapid remodelling of the whole area; (4) reformation of new arcades and sprouts to replace a destroyed segment of the blastema and subsequent normal growth. This mechanism, compensating for focal damage, is so efficient that no apparent retardation in rate of growth of the entire blastema as seen in the ear chamber occurs below a threshold dose of 2,000 r (single tissue dose) of roentgen or gamma rays[14] beyond which cessation of growth occurs within twenty-four hours after irradiation. This threshold dose of 2,000 r is subject to considerable individual variation for different animals. A small proportion of ear chambers shows unretarded progressive growth after a dose of 3,000 r gamma rays from a Co^{60} source. However, below the threshold level for an individual ear chamber, no proportionate slowing down of linear growth takes place for a blastema 6 mm. in diameter. In these experiments, the observations are restricted to a total normal growth period of not greater than six weeks, as defined by the size of the tissue on the central table. While it is possible that irradiated endothelium may show progressive fall-off in growth rate after irradiation proportional to dosage if the observations were applied to a

larger blastema in order to extend the period of observation, the results obtained fail to support this suggestion. It has been shown previously that the measurement of linear growth of the blastema is essentially a straight line relationship for the blastema growing to completion[15]—a finding which has been presented in support of the hypothesis that the parameter of linear growth rate depends on the tactical mechanism of cell migration as opposed to cell division. The findings which have been presented here, together with previous quantitative studies,[14,15] support the idea that irradiation of a capillary sprout has a direct all-or-none effect on migration of the growing tip. This effect may be temporary, followed by recovery and migratory extension at an unreduced rate; it may be permanent as a result of degeneration of the bud or retraction into parent arcades, or there may be *an apparent reduction in the capacity for migratory growth* in the presence of an altered cellular environment (*viz: fibrous barrier*). That the latter reduction in rate of growth is not due to an intrinsic radiation induced change in the vascular endothelium is shown by the phenomenon of *super-regeneration* which has been described and illustrated. Liberation of such irradiated sprouts and vessels from the "fibrous barrier environment" by manipulative trauma and the substitution of a focal hemorrhagic medium result in uninhibited migratory growth, which reverts from the nonhemorrhagic budding and looping type of advance to the florid hemorrhagic sprout formation. Such super-regeneration can be induced at will in irradiated ear chamber tissues, which have received single tissue doses of up to 5,000 r gamma rays—a dose which in itself, jeopardizes the viability of the entire tissue enclosed by the chamber, and which administered to areas of rabbit ear skin of comparable size results in acute radionecrosis in a considerable percentage of animals. The latent period between threshold irradiation and the induction of super-regeneration in the static regenerate has not been quantitatively determined, but a successful manipulation has been performed as early as ten days after irradiation with 3,000 r. Once the secondary radiation reaction has subsided in the chamber tissues, *i.e.*, approximately four weeks after irradiation[16] and provided the blastema has not degenerated *in toto* (acute radionecrosis), super-regeneration can be induced readily, and in the absence of infection or mechanical faults in the chamber tolerances, the blastema will grow and cover the table area at an even linear rate of growth, comparable to that which existed prior to irradiation within the limits of experimental accuracy. On occasion secondary thrombosis in the irradiated tissues causes partial or complete destruction of the blastema. In cases of partial destruction, the avascular infarct acts as a fibrous barrier and is subject to the same conditions of vascularization (slow bud and loop formation, or induced super-regeneration) as attends radiation induced fibrous barriers. That the slow nonhemorrhagic vascularization of ischemic residue is not due to an intrinsic endothelial deficiency caused by irradiation is shown by: (1) normal hemorrhagic sprouting, which may take place elsewhere but concurrently in the irradiated regenerate (Fig. 11 and 12) *vide supra;* (2) focal aberrant, concentrated sprout formations (Fig. 5*a* and 8*a*); (3) the induction of super-regeneration and; (4) a parallel type of vascularization seen in nonirradiated tissues, *e.g.*, tubercles.[4]

The term, *super-regeneration* has been adopted from a usage by Litschko[6] to define a radiation phenomenon he observed, which is considered to resemble closely that described here. Litschko found that irradiation of the stump resulting from amputation of a limb in the axolotl prevented regeneration of a new appendage. He found that the irradiated stump contained avascular fibrous tissue which on removal (secondary amputation) permitted the normal regeneration of a new limb—a phenomenon he termed *super-regeneration*. It is not en-

tirely clear whether Litschko's secondary amputation transected irradiated tissue, or whether super-regeneration originated in unirradiated tissue. In the results presented here, continuous *in vivo* observation has shown that vascular super-regeneration definitely occurs within the irradiated tissue and from the irradiated endothelial cells as an epimorphic process.

The formation of a *fibrous barrier* follows a clear pattern of events. A threshold level of irradiation causes temporary inhibition of vascular sprouting and destruction, sufficiently widespread to prevent effective compensation by competent vascular zones (*vide supra*). The irradiation also causes cessation of the gross hemorrhagic diapedesis through the sprouts, and no further addition to the hemorrhagic zone. Concurrently, macrophages already recruited from the monocyte influx,[5] together with further systemic recruitment of monocyte-macrophage cell systems after irradiation, provide active demolition of the hemorrhagic zone, and considerable fibroblast migration and differentiation continues. This results in a mature ischemic fibrous barrier zone which modifies further vascularization despite endothelial recovery and the capacity for growth. When a relative ischemia and fibrosis is uniformly distributed in tissues (*e.g.*, radiation atrophy), while small focal injuries may be followed by adequate repair (super-regeneration), a generalized change (*e.g.*, inflammation) requiring rapid extensive remodelling and growth of vascular endothelium to maintain tissue viability is often followed by necrosis (*i.e.*, chronic radionecrosis). This is not so much due to intrinsic incompetence of individual cells but results from a qualitative change in form of the entire tissue. In tissues where fibrosis is naturally or artificially excessive, vascular lability is indirectly impaired. The disastrous results which may follow inflammations in tendons and joints or in atrophic conditions (*e.g.*, radiation atrophy and scarring) point to the apparent cause. Whether fibrosis acts by mechanical or chemical means in impairing vascular remodelling or regeneration is uncertain. The inhibition of epithelial migration by beryllium salts with the formation of a "dermal seal"[13] seems a similar phenomenon. Here, the changes have been interpreted as resulting from an inhibition of de-differentiation of epithelium, presumably from chemical action. The morphologic changes observed in vascular sprouts in irradiation induced fibrous barriers suggest that de-differentiation still takes place in endothelium with aberrant sprout formation, but migration is ineffectual due to mechanical barriers.

Finally, it may be concluded that regenerating vascular endothelium, despite its wide spectrum of radiosensitivity *in vivo*, posseses great powers of recovery from radiation damage. However, such powers of recovery may be masked by concurrent qualitative changes in the regenerate, particularly in the endoskeletal component (fibrillogenesis), but recovery and competence of vascular endothelium can be revealed by the induction of super-regeneration. It is also clear that the behavior of irradiated cells in an ideal *in vitro* system may provide striking differences from that seen *in vivo* despite facilities for nutrition and excretion in the latter, which also seem ideal. The growing vascular sprouts and arcades are provided with a free circulation of blood and optimal conditions for diffusion of nutrient and waste products. Also, the high oxygen tension present favors radiation damage. The comparison of this system, with an ideal *in vitro* system such as that achieved by Puck and Marcus,[9] in which a logarithmic growth of mammalian cells obtains, shows great differences in response to irradiation. The indirect factors which operate *in vivo* are clearly not a simple matter of competition for nutrients, but cellular behavior is largely determined by the *form* of the tissue in question. An interpretation of the performance of regenerating cellular systems *in vivo* in terms of population number or size is strictly lim-

ited and an unreal index of tissue survival or adaptation of the organism. The *tactical* aspect of growth emphasized by Medawar[7] and embodied in the studies of Thompson[12] on tissue form has a clear application, particularly in assessing the effect of substances and agents which modify growth. As Medawar has stressed, "the *tactics* of regeneration as of embryonic development are primarily and fundamentally a matter of movement of cell substance, cells and cell groups." In the final analysis, repair of injury in mammalian tissues depends on the successful growth of an endothelial sprout or segment. That the tactics of this mechanism, as in epithelial growth *in vivo* are one of migration is clear. While agents which modify number of cell population and synthesis of protoplasm may determine survival of a tissue *per se*, repair of injury and tissue restoration often accomplished as an initial event without substantial synthesis of new cells, whether the morphologic change is predominantly one of morphallaxis or epimorphosis. An agent (*e.g.*, ionizing radiations) which causes death of a proportion of cells and reduces the supply of new cells and leads to an atrophic tissue state does not necessarily compromise the efficiency of repair as an immediate event. In wounds of the integument, successful healing by no means depends on the volumetric amount of granulation tissue synthesized. In fact, overproduction of granulation tissue may impair restoration of tissue continuity as in burns, hypertrophic scars and keloids. The reduction of granulation tissue by irradiation as a therapeutic measure has been used to facilitate healing of wounds of this type. It is also known that radiation burns (necrosis) may be followed by subsequent keloid development in the area of necrosis. These findings suggest that the tactical competence of viable repair tissues after irradiation remains intrinsically unimpaired; while irradiation may inhibit synthetic growth of endothelium, recovery does occur and is apparently complete. Such modification of regenerative growth as does take place following injury depends on indirect factors. Fibrillogenesis which precedes the atrophic tissue state is particularly important in this respect.

We may ask what factors operate in delayed radionecrosis. Such tissue is atrophic, the vascular index is reduced, and a quantitative enhancement of fibrillogenesis in the connective tissue has resulted in an excess of collagen deposition, often accompanied by pathologic hyalinization of the fibers. Strictly focal injuries in such tissue, unaccompanied by spreading infection, are adequately repaired by super-regeneration. Diffuse injuries (knocks, contusions) may still be repaired, similarly, by super-regeneration if scattered focal damage results, insufficient to require extensive vascular remodelling of the whole tissue. However, most diffuse injuries and inflammations of necessity are accompanied by generalized hyperemia and vascular remodelling for which unimpeded lability of vascular endothelium is essential. Also the reduced vascular and connective tissue cell concentration of atrophic tissues reduces the surface area of the reparative front. Again, a quantitative increase in the noncellular endoskeleton, particularly collagen, together with irreversible changes in such collagen (hyalinization) not only acts as a fibrous barrier to tactical growth, but as foreign material prolonging the demolition period of repair, often beyond superable limits. Vascular regeneration in such circumstances is intrinsically competent but abortive, due to these overriding factors, and complete necrosis or at best partial necrosis accompanied by chronic inflammation results. The ever present risk of superadded infection magnifies the hazard to vascular regeneration and is often the precipitating cause of delayed radionecrosis. The experimental studies which have been presented in this paper clearly indicate, that intrinsic defects in surviving cell fraction presumed to result from the exposure to ionizing radiation are not responsible for

inadequate repair, subsequently in response to injury. Indirect factors, *viz.*, changes in tissue form, altered cellular indices, fibrillogenesis and irreversible changes (hyalinization) in the stroma, must be implemented, and are nonspecific in character and common to all atrophic connective tissues. In accounting for poor healing of irradiated tissues, one must suspect the validity of such conceptions as "failure to de-differentiate," "premature aging," "incomplete recovery" and "somatic mutation," all of which imply altered cell characteristics which should be demonstrable *in vitro* but which have no proved basis *in vivo*.

SUMMARY

1. The morphologic characteristics of regenerating blood capillaries (sprouting, budding and looping) are described as observed in the living state in rabbit ear chambers.

2. The effects of ionizing radiations (gamma rays from Co^{60} and 200 kv. roentgen rays) on capillary regeneration are described and illustrated.

3. Attention is drawn to the indirect effects of fibrillogenesis ("fibrous barrier formation") on capillary regeneration and remodelling in both irradiated and nonirradiated tissues.

4. Fibrillogenesis inhibits vascular regeneration and modifies morphologic appearances, despite an intrinsic competence for growth of the irradiated vascular component. The morphologic events which follow irradiation of growing repair tissues to high dosage and result in cessation of growth and in the formation of a "fibrous barrier" are described and illustrated.

5. The phenomenon, which has been termed "*super-regeneration*," is described and illustrated. This phenomenon shows that the intrinsic competence for growth of irradiated endothelium remains essentially intact despite cessation of such growth in a repair blastema, irradiated to a sufficient (threshold) level of dosage.

6. The implications of these findings are discussed in relation to radionecrosis, radiosensitivity of cell systems and the comparisons to be drawn from growth of simple cell systems *in vitro* in respect to integrated growth *in vivo*.

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EDITORIALS

INTRAVENOUS ANGIOCARDIOGRAPHY: THE TWENTIETH YEAR

THE practical method of visualizing the four cardiac chambers and great vessels was reported by Robb and Steinberg in 1938, and published in this JOURNAL in January, 1939, just twenty years ago. Nowadays, when there is a growing tendency to insert needles and introduce catheters into the heart and great vessels, it may seem archaic to discuss the method of intravenous angiocardiography.

Failure to completely visualize the cardiovascular system by intravenous angiocardiography occurs because the technique requires meticulous attention to a few details which have, incidentally, remained unchanged during the twenty year period. A large special bore (12 gauge) needle-stopcock unit and syringe are necessary for rapid injection. Speed of injection is essential if the contrast medium is to be delivered into the heart as a bolus. Moderate inspiration during injection aids in sucking the contrast material into the thorax; keeping the patient from inadvertently performing the Valsalva maneuver prevents delay of passage of the medium into the superior vena cava. In cooperative adults, the erect position with thorax against cassette minimizes the possibility of the cardiovascular structures being obscured by the thoracic bony cage and diaphragm.

Advances in rapid serial roentgenography (multiple exposures per second and cineangiocardiography with the image amplifier) have made unnecessary preliminary circulation time determinations in patients without heart failure and/or valvular heart disease. Roentgen exposures of five seconds in infants and a duration of eight seconds in adults are all that are needed for a complete study of the passage of contrast material from the arm into the thoracic aorta. In heart failure and in patients with valvular heart disease (especially mitral valvular disease), a modified circulation time evidenced by a bitter taste can be determined by rapidly injecting 3 cc. sodium decholin mixed with 15 cc. normal saline via the Robb-Steinberg needle. In patients with a prolonged circulation time, multiple exposures per second are unnecessary because the slowed circulation results in good visualization of the cardiovascular system. Roentgenograms made at one second intervals for the duration of the circulation time have proved very satisfactory.

Improvements in the concentrated organic iodide contrast materials have reduced reactions during angiocardiography. Sodium acetrizoate (urokon) 70 per cent is a highly satisfactory agent. Despite inability of predicting reactions by pretesting (intradermal, intraoral, ophthalmic, and intravenous tests), the number of untoward reactions remains remarkably small. Recent observations have also shown that a calm, confident, and nonapprehensive attitude during injection with care not to alarm the patient still further reduces reactions. In many centers experience with large numbers of cases has shown that preliminary medication to allay emotional stress and the administration of antihistamines are unnecessary. Anesthesia, unless the patient is uncooperative, should be avoided because such agents by themselves are hazardous. Although uncommon, reactions such as hypotension, syncope, shock, apnea, pulmonary edema, cardiac arrest, fever and convulsions still occur; fatalities,

however, are rare. These reactions are probably due to drug idiosyncrasy and may be minimized by exercising good judgment in the selection of the case for angiocardiography. The high risk patients (cyanotic heart disease cases) should be studied only in the hospital where special facilities and skills are available. If cardiac arrest occurs, open thoracotomy and cardiac massage followed by proper anesthesia and surgical closure of the chest may be life-saving. Search for the ideal, safe and nonreactive contrast substance must continue.

Intravenous angiocardiography, like right heart catheterization, because of the complexity of some types of heart disease, cannot be expected to be suitable for all types of disease of the cardiovascular system. Accordingly, selective angiography by needle and catheter injection, particularly for the demonstration of valvular deformities and certain types of shunts, may be necessary. Like all new special procedures, their indications need to be evaluated. However, it seems unlikely that these techniques will displace intravenous angiocardiography.

Among the striking achievements with intravenous angiocardiography and which may also serve as an indication for the procedure are: the differential diagnosis of mediastinal tumor and aneurysm; the diagnosis of pericardial effusion and differentiation from a dilated and enlarged heart; the diagnosis of intracavitary and intrapericardial tumors; the diagnosis of myxoma of the atria and atrial thrombi; pulmonary arterial and venous circulatory abnormalities (congenital and acquired), especially those due to pulmonary disease; the demonstration of pulmonary arteriovenous fistulas; and aortic anomalies ranging from aneurysm and hypoplasia to coarctation. In cyanotic congenital heart disease, the demonstration of right-to-left shunts (tetralogy of Fallot, tricuspid atresia and reversed ductus arteriosus) is one of the most important reasons for angiocardiography. In cancer of the lung, the determination of great vessel obstruction and occlusion is of undeniable benefit in establishing resectability. Nephrotomography, developed by Dr. John A. Evans and his associates at The New York Hospital, utilizes the Robb-Steinberg method to differentiate between renal cysts and neoplasms.

The prime achievement of intravenous angiocardiography is that it ushered in the era of safe contrast visualization of the cardiovascular and pulmonary systems, making cardiovascular anatomy discernible during life. As a result, roentgen interpretation and diagnosis of disease of the cardiopulmonary system reached a degree of accuracy previously thought impossible. Obviously, by enhancing the preoperative diagnosis of cardiovascular abnormalities, the surgical repair of such defects, when indicated, is aided. Intravenous angiocardiography because of its simplicity and relative freedom from severe reactions remains the method of choice for visualization of many of the cardiovascular and pulmonary structures.

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HARRY M. WEBER
1899–1958

ON October 4, 1958, the world of radiology lost one of its most distinguished teachers and leaders when Harry M. Weber died unexpectedly after a brief illness. Many radiologists the world over will feel the loss of this man who was such a steadfast friend.

Harry Weber was born June 30, 1899, in Lewistown, Montana. He was educated at St. John's University, Collegeville, Minnesota, and the University of Minnesota. The latter institution granted him the degrees of Bachelor of Science in 1924 and Doctor of Medicine in 1926. After a period of internship in St. Joseph's and Ancker Hospitals in St. Paul, he became physician in a construction camp at Berne, Washington. In January, 1927, he entered the Mayo

Foundation as a fellow in radiology where his preceptors were Drs. B. R. Kirklin, A. B. Moore and C. G. Sutherland. It was during this period of training that he became interested in roentgenologic diagnosis of diseases of the large intestine, an interest that culminated in 1930 with the publication of his first paper dealing with the double-contrast method for examining the colon.

In October, 1931, he joined the staff of the Mayo Clinic as a member of the section of diagnostic roentgenology. He continued his interest in the diagnosis of diseases of the intestines and soon became known as an authority in the field. Largely through his efforts, the roentgenologic diagnosis of diseases of the large intestine was developed and improved and roentgenologists the world over began to look to him for advice concerning techniques of examination and interpretation.

In July, 1951, he became head of the section of diagnostic roentgenology of the Mayo Clinic and in 1954 was advanced to the rank of professor of radiology in the Mayo Foundation for Medical Education and Research, a division of the Graduate School of the University of Minnesota.

During the course of his eminent career, Harry Weber contributed many articles to the radiologic literature. Most of these were concerned with the diagnosis of diseases of the gastrointestinal tract. He was a superb fluoroscopist, a careful observer and an accurate analyst. His conclusions were carefully thought out and written in precise prose. He was a perfectionist in his choice of words, often consulting Greek and Latin dictionaries before finding the exact word for a specific meaning. Only a few of his associates know of the several papers he prepared but refused to submit for publication because of his dissatisfaction either with the conclusions or with the style of writing.

He possessed an adequate command of German, a language he inherited from his parents. On several occasions in 1938, while visiting in Germany, he addressed meetings of radiologic societies in their native language. He read the German literature in his field of interest as often as he read the English.

Perhaps Harry Weber's greatest accomplishment in radiology was as a teacher. Many of the leaders in the roentgenologic diagnosis of intestinal disease learned their fundamentals from him, either while working and studying with him, or by listening to his lectures and courses of instruction given at meetings of scientific societies. Besides being responsible for the training of graduate students in radiology at the Mayo Foundation, he was also, for ten years, director of instructional courses of the American Roentgen Ray Society.

Harry Weber gave liberally of himself and his time to organized medicine. He was elected president of the Minnesota Radiological Society in 1940, chairman of the section of radiology of the American Medical Association in 1948 and president of the American Roentgen Ray Society in 1953. In addition to serving as director of instructional courses for the latter organization he had also, at one time or another, been a member of its publication committee and its executive council, serving a year as chairman of the latter. In 1951 he was consultant to the committee on x-ray department planning of the American College of Radiology and in 1949 was co-chairman of the historical committee of the Minnesota State Medical Association. For many years he served as delegate to this latter organization, representing his county medical society.

In 1934 he was certified as a specialist in roentgenology by the American Board of Radiology. He was a fellow of the American College of Radiology and a member of the Minnesota State Medical Association, the Zumbro Valley Medical Society, the Southern Minnesota Medical Association, the American Medical Association, the Minnesota Radiological Society, the American Roentgen Ray Society, the Radiological Society of North America, the Rocky Mountain Radiological Society and the

American Gastro-Enterological Association. He was an honorary member of the Detroit Roentgen Ray and Radium Society, the St. Louis Medical Society and the Society of Radiology and Physical Therapy of Havana, Cuba. He was a member of Sigma Chi, Phi Rho Sigma and the Society of Sigma Xi.

In 1937 he received the first award of the Fifth International Congress of Radiology for the excellence of his scientific exhibit, and in 1940 an exhibit on polyps of the colon and rectum that he helped prepare won a silver medal at the meeting of the American Medical Association. He was the recipient of several certificates of merit from the American Roentgen Ray Society. He had given the Russell D. Carman Memorial Lecture of the St. Louis Radiological Society in 1948 and was chosen to give the first Fred I. Hodges Society Lecture at the University of Michigan on April 11, 1958.

A few days after the attack on Pearl Harbor, Harry Weber was in the uniform of a lieutenant commander of the United States Navy. He was assigned to active duty at the Naval Hospital at Corona, California, where he was made responsible for organizing and equipping the x-ray department. He returned to civilian life in July, 1942, at which time he suffered the first signs of the coronary heart disease which, more than sixteen years later, was to take his life so unexpectedly. Nevertheless, after recovering from the initial attack, he continued to live a normal, active, happy life; he showed no signs of fearing the ultimate, a fact that buoyed up several of his friends who suffered from similar conditions.

On February 16, 1937, Harry Weber was married to Kathleen Osborn. The couple had three sons, Harry Osborn, a senior at the University of Minnesota, Frederic William, a student at the University of Notre Dame, and George Osborn, a student at St. John's School, Collegeville, Minnesota. Besides his immediate family he is survived by his mother, Mrs. Matt Weber of Hastings, Minnesota, and a sister, Mrs. B. Marden Black of Rochester, Minnesota.

Harry was interested in sports, especially football and golf. During the last twenty years of his life, he became an ardent golfer, often enjoying a few holes in the company of his colleagues after the work of the day was over. In 1956, he won the Manges Trophy at the meeting of the American Roentgen Ray Society. He had served as president of the Rochester Golf and Country Club. He was instrumental in organizing a yearly golf tournament for the staff and fellows of his department at the Mayo Clinic and he participated in it annually until prevented in 1958 by the attack of pneumonia which precipitated his final illness.

Harry was modest, friendly, kind, religious and genuinely interested in the welfare of his patients, his students and his friends. One of these has written, "From the flood of memories and emotions which engulf me every day as I look up from my desk to see Harry's picture, the most insistent thoughts which come to mind are those which relate to Harry's enormous capacity for steadfast friendship and his sincere and never failing modesty in evaluating his own abilities and importance. I think I have known few people more sincere in this regard."

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HIGDON BRYANT ELKINS
1907–1958

DR. HIGDON B. ELKINS, Professor of Radiology in charge of Radiation Therapy at the State University of Iowa College of Medicine, died November 29, 1958, at the age of fifty-one. Dr. Elkins had suffered a severe coronary occlusion in April, 1958, and never recovered sufficiently to resume more than part-time activities. His death was attributed to pulmonary embolism.

Dr. Elkins was born in Purdy, Missouri, May 26, 1907. He received his bachelor's degree in 1928 and his degree of Doctor of Medicine in 1930 from Washington Uni-

versity in St. Louis. Following an internship at St. Luke's Hospital, St. Louis, he became a resident in radiology at the University of Iowa. He practiced in Ely, Nevada, from 1934 to 1942, and it was during this time that he developed the skill and that sense of being a real physician that was to mean so much to him and to his patients in the practice of therapeutic radiology. From 1942 to 1946, he served in the army, spending considerable time in the European theater of the war and was discharged as a major. On return to civilian life, he felt the need of further training in radiology and returned to the State University of Iowa in 1946 as a Fellow. From then, his rise was rapid at this institution; he became Assistant Professor in 1947, Associate Professor in 1950, and full Professor in 1956.

"H.B.," as he was known to many of his intimate friends, was a fortunate combination of an excellent radiation therapist and an inspiring teacher. He was also exceptionally well grounded in the physical aspects of his specialty. Moreover, he was a physician whose first consideration was the care of his patients. His broad background in the practice of medicine gave him an insight into general medical problems which is not often attained by those trained in the narrow field of a single specialty. It enabled him to discuss the problems of his patients on equal terms with consultants from medicine, surgery, or the other specialties. Only those who knew Dr. Elkins well realized the thought that went into the planning of the care of his patients. And his patients thought of him and knew him as a friend as well as their physician.

Scientific radiology has been advanced by the many contributions from his pen. He was in demand as a speaker at conferences and national meetings, wherever radiation therapy was discussed, and he was particularly interested in the relatively new field of radioisotope application to therapeutic problems. The American Board of Radiology recognized his excellence and invited him as a guest examiner in radiotherapy and nuclear medicine on many occasions.

His membership in national scientific societies included the American Medical Association and its constituent groups; also, the American Roentgen Ray Society, the Radiological Society of North America, the American Radium Society, and the Society of Nuclear Medicine of which he was a charter member. He was a Fellow of the American College of Radiology and, of course, a diplomate of the American Board of Radiology.

Dr. Elkins is survived by his wife, the former Katharine Eberhardt of Salt Lake City, and by his mother. His place will be difficult to fill. He will be sorely missed by his many friends in his home community and throughout the country. One friend spoke for many, "We all looked to him for advice numerous times and always, no matter how busy he was, he found time to be gracious and helpful." Such was the man we mourn!

H. Dabney Kerr, M.D.

SOCIETY PROCEEDINGS AND NEWS ITEMS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. C. Allen Good, Mayo Clinic, Rochester, Minn. Annual meeting: Netherland-Hilton Hotel, Cincinnati, Ohio, Sept. 22–25, 1959.

American Radium Society
Secretary, Dr. Robert L. Brown, P.O. Box 459, Emory University, Ga. Annual meeting: To be announced.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 713 E. Genesee St., Syracuse 2, N. Y. Annual meeting: Palmer House, Chicago, Ill., Nov. 15–20, 1959.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Illinois. Annual meeting: Feb. 3–6, 1960, Roosevelt Hotel, New Orleans, La.

Section on Radiology, American Medical Association
Secretary, Dr. T. Leucutia, Harper Hospital, Detroit 1, Mich. Annual meeting: June 8–12, 1959, Atlantic City, N. J.

American Board of Radiology
Secretary, Dr. H. Dabney Kerr. Correspondence should be directed to Kahler Hotel Building, Rochester, Minn. The Board will conduct an examination June 2–4, 1959, Palmer House, Chicago. The deadline for filing applications for this examination was February 15, 1959. If sufficient interest is shown, a special examination will be offered at this time in Nuclear Medicine to diplomates in Radiology or Therapeutic Radiology. The deadline for filing applications was April 1, 1959.

Ninth International Congress of Radiology
Secretary-General, Dr. Hans v. Braunbehrens. Request for information should be addressed to Dr. Viktor Loeck, Kongressbüro, Forsthausstrasse 76, Frankfurt am Main, Germany. Meets in Munich, Germany, July 23 to July 30, 1959.

Seventh Inter-American Congress of Radiology
Secretary, Dr. Laura Fariñas, Havana, Cuba. *Counselor for the United States*, Dr. J. A. del Regato, Penrose Cancer Hospital, 2200 North Cascade Avenue, Colorado Springs, Colorado. The meeting will be held in Brazil in 1961.

Alabama Radiological Society
Secretary, Dr. J. A. Meadows, Jr., Medical Arts Bldg., Birmingham 5, Ala. Meets time and place Alabama State Medical Association.

American Nuclear Society
Executive-Secretary, Octave J. Du Temple, 86 E. Randolph St., Chicago, Ill.

Arizona Radiological Society
Secretary, Dr. R. Lee Foster, 1313 N. Second St., Phoenix, Ariz. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Arkansas Radiological Society
Secretary, Dr. E. A. Mendelsohn, Holt-Krock Clinic, Fort Smith, Ark. Meets every three months and also at time and place of State Medical Association.

Association of University Radiologists
Secretary, Dr. Paul Riemenschneider, Medical College. State University of New York, Syracuse, N. Y. Annual meeting: Spring, 1959 in Atlanta, Ga.

Atlanta Radiological Society
Secretary, Dr. J. L. Clements, Jr., 35 Linden Ave., N.E., Atlanta 8, Ga. Meets monthly, except during three summer months, on second Friday evening.

Blockly Radiological Society
Secretary, Dr. Samuel Finkelman, 101 S. Twentieth St. Philadelphia, Pa.

Brooklyn Radiological Society
Secretary, Dr. George A. Manfredonia, One Hanson Place, Brooklyn, N. Y. Meets first Thursday of each month October through May.

Buffalo Radiological Society
Secretary, Dr. Alfred Dobrak, 710 Leydecker Rd., Buffalo 24, N. Y. Meets second Monday evening each month, October to May inclusive.

Central New York Radiological Society
Secretary, Dr. Joseph A. Head, 150 Marshall St., Syracuse, N. Y. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary, Dr. William B. Schwartz, 1500 W. Third Ave., Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, November, January, March and May at Fort Hayes Hotel, Columbus, Ohio.

Central Society of Nuclear Medicine
Secretary, Dr. Robert S. Landauer, Radiation Center Building, 1903 West Harrison St., Chicago 12, Ill.

Chicago Roentgen Society
Secretary, Dr. George B. Cahill, 802 Burns Ave., Flossmoor, Ill. Meets second Thursday of each month, October to April except December at the Sheraton Hotel at 8:00 P.M.

Cleveland Radiological Society
Secretary, Dr. Frederick A. Rose, 2065 Adelbert Rd., Cleveland 6, Ohio. Meetings at 7:00 P.M. on fourth Monday of each month from October to April at Tudor Arms Hotel

Colorado Radiological Society
Secretary, Dr. Lorenz R. Wurtzebach, 601 E. Nineteenth Ave., Denver, Colo. Meets third Friday of each month at Denver Athletic Club.

Connecticut Valley Radiologic Society
Secretary, Dr. Paul J. Kingston, 114 Woodland St., Hartford, Conn. Meets first Friday in February and April.

Dallas-Fort Worth Radiological Club
Secretary, Dr. A. H. Keene, 3707 Gaston Ave., Dallas, Texas. Meets monthly, third Monday, at Greater Fort Worth International Airport at 6:30 P.M.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. Joseph O. Reed, Jr., Harper Hospital, Detroit 1, Mich. Meets monthly first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

East Bay Roentgen Society
Secretary, Dr. Dan Tucker, 434 30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

East Tennessee Radiological Society
Secretary, Dr. Charles W. Reavis, Baroness Erlanger Hospital, Chattanooga, Tenn. Meets in January and September.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Eastern Radiological Society
Secretary, Dr. John D. Osmond, Jr., Euclid-Glenville Hospital, Cleveland 19, Ohio.

Florida Radiological Society
Secretary, Dr. Russell D. D. Hoover, 49th St., West Palm Beach, Fla. Meets twice annually, in the spring with the annual State Society meeting, and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. Joseph C. Rush, 1501 Jungle, St. Petersburg, Fla.

Georgia Radiological Society
Secretary, Dr. B. H. Malone, 1406 Reynolds St., Brunswick, Ga. Meets Spring and Fall with Annual State Society Meeting.

Greater Miami Radiological Society
Secretary, Dr. George P. Daurelle, Jackson Memorial Hospital, Miami 36, Fla. Meets monthly third Wednesday at 8 P.M. at Jackson Memorial Hospital, Miami, Fla.

Greater St. Louis Radiological Society
Secretary, Dr. Charles J. Cherre, 462 N. Taylor, St. Louis 8, Mo.

Houston Radiological Society
Secretary, Dr. Edward B. Singleton, 6621 Fannin St., Houston 25, Texas. Meets last Monday each month, Seminar Room, Doctors' Club of Houston.

Idaho State Radiological Society
Secretary, Dr. Charles R. McWilliams, Magic Valley Memorial Hospital, Twin Falls, Idaho. Meets annually at same time as Idaho State Medical Association.

Illinois Radiological Society
Secretary, Dr. Stephen L. Casper, 1101 Maine St., Quincy, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. C. A. Stayton, Jr., 313 Hume-Mansur Bldg., Indianapolis 4, Ind. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

Iowa Radiological Society
Secretary, Dr. James T. McMillan, 1104 Bankers Trust Bldg., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society
Secretary, Dr. Lewis G. Allen, 807 Huron Bldg., Kansas City, Kansas. Meets in spring with State Medical Society, and in winter on call.

Kentucky Radiological Society
Secretary, Dr. Robert H. Akers, V. A. Hospital, Louisville 2, Ky. Meets monthly on second Friday at Seelbach Hotel, Louisville.

Kings County Radiological Society
Secretary, Dr. C. P. Naidorf, 411 Parkside Ave. Brooklyn 26, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Los Angeles Radiological Society
Secretary, Dr. Robert B. Engle, St. Luke Hospital, Pasadena, Calif. Meets second Wednesday of month in September, November, March, April and June at Los Angeles County Medical Association Building, Los Angeles.

Maine Radiological Society
Secretary, Dr. Francis J. O'Connor, Augusta General Hospital, Augusta, Maine. Meets in June, September, December and March.

Maryland Radiological Society
Secretary, Dr. Nathan B. Hyman, 1805 Eutaw Place, Baltimore 17, Md.

Memphis Roentgen Society
Secretary, Dr. James L. Booth, 899 Madison Ave., Memphis 3, Tenn. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society
Secretary, Dr. S. F. Johnson, 2197 Los Arrow Dr., Dayton 9, Ohio. Meets second Friday of fall and winter months.

Mid-Hudson Radiological Society
Secretary, Dr. Joseph Sorrentino, St. Francis Hospital, Poughkeepsie, N. Y. Meets 8:30 P.M., fourth Wednesday each month, September to May.

Milwaukee Roentgen Ray Society
Secretary, Dr. Joseph F. Wepfer, 5000 W. Chambers St., Milwaukee 10, Wis. Meets monthly on fourth Monday at University Club.

Minnesota Radiological Society
Secretary, Dr. Donald H. Peterson, 853 Medical Arts Bldg., Minneapolis 2, Minn. Meets three times annually, in fall, winter and spring.

Mississippi Radiological Society
Secretary, Dr. Bernard T. Hickman, University Medical Center, Jackson, Miss. Meets third Thursday of each month at Hotel Edwards, Jackson, at 6:00 P.M.

Montana Radiological Society
Secretary, Dr. Arthur T. Austin, 104 The Doctors Bldg., Billings, Montana. Meets at least once a year.

Nassau Radiological Society
Secretary, Dr. Jerome Zwanger, 126 Hicksville Rd., Massapequa, L. I., N. Y. Meets second Tuesday of the month in February, April, June, October and December.

Nebraska Radiological Society
Secretary, Dr. Warren Q. Bradley, 924 Sharp Bldg., Lincoln, Nebraska. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

New England Roentgen Ray Society
Secretary, Dr. John E. Gary, 1180 Beacon St., Brookline 46, Mass. Meets third Friday of each month, October through May, at The Longwood Towers, Brookline, Mass.

New Hampshire Roentgen Ray Society
Secretary, Dr. Paul Y. Hasserjian, 1470 Elm St., Manchester, N. H. Meets four to six times yearly.

New York Roentgen Society
Secretary, Dr. Harold G. Jacobson, 210th St. and Bainbridge Ave., New York 67, N. Y. Meets monthly on third Monday, New York Academy of Medicine, at 4:30 P.M.

North Carolina Radiological Society
Secretary, Dr. Charles A. Bream, 211 McCauley St., Chapel Hill, N. C. Meets in May and October.

North Dakota Radiological Society
Secretary, Dr. Marianne Wallis, St. Joseph's Hospital, Minot, N. D. Meetings by announcement.

North Florida Radiological Society
Secretary, Dr. Paul A. Mori, 800 Miami Road, Jacksonville 7, Fla. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
Secretary, Dr. Irving Van Woert, Jr., Albany Hospital, Albany, N. Y. Meets in Albany area on second Wednesday of October, November, March and April.

Northern California Radiological Society
Secretary, Dr. Rob H. Kirkpatrick, 1219 28th St., Sacramento, Calif. Meets at dinner last Monday of each month, September to June.

Ohio State Radiological Society
Secretary, Dr. Francis C. Curtzwiler, 421 Michigan St., Toledo 2, Ohio. Annual meeting: Cincinnati, Ohio, May, 1959.

Oklahoma State Radiological Society
Secretary, Dr. Edmond H. Kalmon, Jr., 300 Northwest 12th Street, Oklahoma City, Okla. Three regular meetings annually.

Oregon Radiological Society
Secretary, Dr. C. V. Allen, Veterans Hospital, Portland, Ore. Meets monthly from October to June on the second Wednesday of each month at 8:00 P.M. at the University Club.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. J. Arch Colbrunn, 19 Royal Oak Drive, Roseburg, Ore. Annual meeting: Seattle, Wash., May 8–10, 1959.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco 8, Calif. Meets annually during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. Walter P. Bitner, 234 State St., Harrisburg, Pa.

Philadelphia Roentgen Ray Society
Secretary, Dr. Roderick L. Tondreau, 3400 Spruce St., Philadelphia 4, Pa. Meets first Thursday of each month, at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Erwin Beck, 3500 Fifth Ave., Pittsburgh 13, Pa. Meets second Wednesday of month, October through June at Park Schenley Restaurant.

Radiological Section, Baltimore Medical Society
Secretary, Dr. James K. V. Willson, 1100 N. Charles St., Baltimore 1, Md. Meets third Tuesday each month, September to May, inclusive.

Radiological Society of Greater Cincinnati
Secretary, Dr. John E. Singer, 468 Doctors' Bldg., Cincinnati 2, Ohio. Meets monthly from September to May on first Monday of each month at 7:30 P.M. at the Cincinnati General Hospital.

Radiological Society of Hawaii
Secretary, Dr. Robert G. Rigler, Straub Clinic, 1020 Kapiolani St., Honolulu 14, Hawaii. Meets third Monday of each month alternately at 4:30 P.M. and 7:30 P.M.

Radiological Society of Greater Kansas City
Secretary, Dr. Samuel B. Chapman, Jr., 830 Argyle Bldg., Kansas City, Mo. Meets last Friday of each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana
Secretary, Dr. Seymour Ochsner, Ochsner Clinic, New Orleans 15, La. Meets annually during Louisiana State Medical Society meeting.

Radiological Society of New Jersey
Secretary, Dr. Andrew P. Dedick, Jr., 67 E. Front St., Red Bank, N. J. Meets at Atlantic City at time of State Medical Society and in November in Newark, N. J.

Radiological Society of New York State
Secretary-Treasurer, Dr Mario C. Gian, 610 Niagara St., Buffalo 1, N. Y. Annual meeting: To be announced.

Radiological Society of South Dakota
Secretary-Treasurer, Dr. Donald J. Peik, 303 S. Minnesota Ave., Sioux Falls, S. D.

Radiological Society of Southern California
Secretary, Dr. Harold P. Tompkins, 658 S. Westlake, Los Angeles 57, Calif.

Redwood Empire Radiological Society
Secretary, Dr. Lee E. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Theodore F. Van Zandt, 501 W. Main St., Rochester 8, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. John H. Freed, 4200 East Ninth Ave., Denver 20, Colo. Annual meeting: Shirley-Savoy Hotel, Denver, Colorado, Aug. 20–22, 1959.

San Antonio-Military Radiological Society
Secretary, Dr. Hugo F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Texas. Meets third Wednesday each month in Fort Sam Houston Officer's Club at 6:30 P.M.

San Diego Radiological Society
Secretary, Dr. Stanley A. Moore, 2466 First Ave., San Diego 1, Calif. Meets first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary, Dr. M. A. Sisson, 450 Sutter St., San Francisco 8, Calif. Meets quarterly at Grison's Steak House.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Connecticut State Medical Society
Secretary, Dr. Ralph J. Littwin, Bristol Hospital, Bristol, Conn. Meetings are held bi-monthly.

Section on Radiology, Medical Society of the District of Columbia
Secretary, Dr. Charles E. Bickham, Jr., 1835 Eye St., N.W., Washington 6, D. C. Meets Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. William Meszaros, 1825 W. Harrison St., Chicago, Ill.

Section on Radiology, Southern Medical Association.
Secretary, Dr. Seymour Ochsner, Ochsner Clinic, 3503 Prytania St., New Orleans 15, La. Annual meeting: Nov. 16–19, 1959, Atlanta, Ga.

Shreveport Radiological Club
Secretary, W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M. September to May inclusive.

Society of Nuclear Medicine
Secretary, Dr. Robert W. Lackey, 452 Metropolitan Bldg., Denver 2, Colo. Annual meeting: June 18–20, 1959, Palmer House, Chicago, Ill.

South Bay Radiological Society
Secretary, Dr. Stanford B. Rossiter, 1111 University Dr., Menlo Park, Calif. Meets second Wednesday of each month.

South Carolina Radiological Society
Secretary, Dr. Wayne Reeser, 804 Burroughs St., Conway, S. C. One meeting with South Carolina Medical Association in May, others arranged by President.

Southern Radiological Conference
Secretary, Dr. Marshall Eskridge, Mobile Infirmary, Mobile, Ala.

Southwestern Radiological Society
Secretary, Dr. Gordon Black, Suite 2-A, El Paso Medical Center, 1501 Arizona, El Paso, Texas. Meets second Tuesday of each month.

Tennessee Radiological Society
Secretary, Dr. James J. Range, P.O. Box 324, Johnson City, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. R. P. O'Bannon, 402 Professional Bldg., 1216 Pennsylvania Ave., Fort Worth 4, Texas. Next meeting January 22 and 23, 1960, Houston, Texas.

Tri-State Radiological Society
Secretary, Dr. James R. Mathews, 118 S. E. First St., Evansville, Ind. Meets last Wednesday of Oct., Jan., March and May, 8:00 P.M. at Elks' Club in Evansville.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society
Secretary, Dr. William R. Christensen, 2033 S. State St., Salt Lake City, Utah. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Virginia Radiological Society
Secretary, Dr. Frank A. Kearney, II, 110 S. Curry St., Phoebus, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Wayne A. Chesledon, 306 Stimson Bldg., Seattle 1, Wash. Meets fourth Monday of each month from September through May at 610 Pine St., Seattle.

West Virginia Radiological Society
Secretary, Dr. Karl J. Myers, 112 N. Woods St., Philippi, W. Va. Meets concurrently with annual meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester Radiological Society
Secretary, Dr. Arnold Myron Wald, 406 Boston Post Road, Port Chester, N. Y. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society
Secretary, Dr. Farrell F. Golden, 5221 Tonyawatha Trail, Madison 4, Wis. Special meetings held during year; annual meeting each spring at Madison, Wis.

X-Ray Study Club of San Francisco
Secretary, Dr. John H. Heald, 450 Sutter St., San Francisco 8, Calif. Meets monthly, third Thursday at 7:30 P.M., Children's Hospital, September through June.

Cuba, Mexico, Puerto Rico and Central America

Asociación de Radiólogos de Centro America y Panamá
Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá, *Secretary-General*, Dr. Roberto Calderón, Calle Central Deste No. 218, Managua, Nicaragua. Meets annually in a rotating manner in the six countries.

Sociedad de Radiología de El Salvador
Secretary, Dr. Rafael Vega Gómez.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad de Radiología y Fisioterapía Cubana
Secretary, Dr. Miguel A. García Plasencia, Hospital Curie, 29 y F, Vedado, Habana, Cuba. Meets monthly at Curie Hospital.

Sociedad Costarricense de Radiologia
Secretary, Dr. James Fernández Carballo, Apartado VIII, San José, Costa Rica.

Sociedad Mexicana de Radiología, A. C.
Calle del Oro No. 15, México 7, D. F.
Secretary-General, Dr. Guillermo Santín. Meets first Monday of each month.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 86, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital, chosen at preceding meeting.

British Empire

Association of Radiologists of the Province of Quebec
Secretary, Dr. Odilon Raymond, 5400 Blvd. Gouin. Quest, Montreal, Que. Meets four times a year.

British Institute of Radiology Incorporated with the Röntgen Society
Honorary Secretary, Dr. John Blewett, 32 Welbeck St., London, W. 1. Meets monthly from October until May.

Faculty of Radiologists
Honorary Secretary, Dr. R. A. Kemp Harper, 47 Lincoln's Inn Fields, London, W.C.2, England. Annual meeting in London (at the Royal College of Surgeons and the Westminster Hospital), June 19–20, 1959.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1.

Canadian Association of Radiologists
Honorary Secretary, Dr. Robert G. Fraser, *Associate Honorary Secretary*, Dr. Jean-Louis Léger, 1555 Summerhill Ave., Montreal 25, Que. Meetings January and June.

Montreal Radiological Study Club
Secretary, Douglas W. MacEwan, 2300 Tupper St., Montreal 25, Quebec. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S. Annual meeting: July 18–25, 1959, Edinburgh.

Société Canadienne-Française d'Electro-Radiologie Médicale
General Secretary, Dr. Louis Ivan Vallée, 1058 rue St-Denis, Montreal 18, Canada. Meets third Saturday each month.

Toronto Radiological Society
Secretary, Dr. L. R. Harnick, Toronto Western Hospital, 399 Bathurst St., Toronto, Ontario. Meets second Monday of each month September through May.

College of Radiologists of Australasia
Honorary Secretary, Dr. E. A. Booth, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W., Australia.

South America

Asociación Argentina de Radiología
Secretary, Dr. Lidio G. Mosca, Avda. Gral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia
Secretary, Dr. Victor A. Añaños, Instituto de Radiologia, Santa Fe 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 P.M. in the Hospital Nacional del Centenario, Santa Fe 1300, Rosario.

O Colégio Brasileiro de Radiologia
Secretary-General, Dr. Walter Bomfins Pontes. Seat: Rua Major Quedinho, 99-2°. Andar, São Paulo, Brazil.

Sociedad Argentina de Radiología, Junta Central, Buenos Aires
Secretary, Dr. Juan L. O. Ambrogna, Montevideo 745, Buenos Aires. Meetings are held monthly.

Sociedad Bolivana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1596, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Nicola Caminha, Av. Mem. de Sa, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644 São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Juan Sabbagh Dacla, Merced 565, Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary, Dr. Alberto Mejia Dzg., Carrera 13, No. 25–31, Apartado aéreo No. 5804, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología y Fisioterapía
Secretary, Dr. Publio Vargas P., Casilla 1242 Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary, Dr. Luis Pinillos Ganoza, Apartado 2306, Lima, Perú. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta 218, Lima.

Sociedad de Radiologia del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiologia.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Ricardo Parada, Av. Agraciada 1464, piso B, Montevideo, Uruguay.

Sociedade de Radiologia de Pernambuco
Secretary, Dr. Manoel Medeiros, Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad Venezolana de Radiología
Secretary-General, Dr. Sebastian Núñez Mier y Terán, Apartado No. 9362, Caracas, Venezuela. Meets monthly third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 256 Chaussée de Wavre, Heverlee-lez-Louvain, Belgium. Meets in February, March, May, June, October, November and December.

Société Française d'Electroradiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris.
Secretary-General, Dr. Ch. Proux, 9, rue Daru, Paris 8e, France.

Ceskoslovensk společnost pro Rëntgenologii a Radiologii v Praze
Secretary, Dr. Roman Bláha, Praha xii, stát. nemocnice, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Deutsche Röntgengesellschaft
Secretary, Professor Dr. med. H. Lossen, Universitäts-Röntgeninstitut. Lagenbeckstr. 1, Mainz, Germany. Annual meeting: To be announced.

Società Italiana di Radiologia Medica e di Medicina Nucleare
Secretary, Dr. Ettore Conte, Ospedale Mauriziano, Torino, Italy. Meets annually.

Nederlandse Vereniging voor Electrologie en Röntgenologie
Secretary, Dr. J. R. von Ronnen, Violenweg 14, den Haag, Netherlands.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Española de Radiologia y Electrologia
Secretary, Dr. D. Aureo Gutierrez Churruca, Esparteros, No. 9, Madrid, Spain. Meets monthly in Madrid.

Schweizerische Gesellschaft für Radiologie und Nuklearmedizin (Société Suisse de Radiologie et de Médecine Nucléaire)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

India

Indian Radiological Association
Secretary, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

The next list of Meetings of Radiological Societies will be published in the July issue of the Journal.

UNIVERSITY OF OKLAHOMA PRESENTS SYMPOSIUM

On May 8 and 9, 1959, the Fifth Annual Surgery, Radiology, Pathology Symposium entitled "Diagnosis and Treatment of Thyroid Diseases" is to be presented by the University of Oklahoma Medical Center, the Oklahoma Association of Pathologists, the Oklahoma State Radiological Society and the Oklahoma Chapter of the American College of Surgeons. For additional information, write the Office of Postgraduate Education, University of Oklahoma Medical Center, 801 Northeast 13th Street, Oklahoma City 4, Oklahoma.

The program has been developed for all physicians with a special interest in the thyroid and its diseases.

COURSE IN RADIOLOGICAL PHYSICS

A course in radiological physics, leading to the degree of Master of Science, is offered under the auspices of the Department of Radiology of the College of Physicians and Surgeons of Columbia University. It is designed to prepare candidates to carry out all the functions of a physicist in a hospital department of radiology; it would also furnish a foundation for those who wish to engage in research in radiological physics. The course includes lectures, seminars, conferences, and laboratory work. Topics included are elementary and advanced radiologic physics, electronics, radiation standardization and protection, radioactive isotopes, biostatistics, radiobiology, and clinical applications of radiation physics. Prerequisite for admission is a bachelor's degree with a major or strong minor in physics, or equivalent scholastic background, and a good academic record. A knowledge of general chemistry and general biology is very desirable.

The course may be given as full-time for one year, or half-time for two, depending on the desires of the class. The total fees amount to about $1,200. For further information write to Dr. Harald H. Rossi, 630 West 168th Street, New York 32, New York.

COURSE TO BE GIVEN IN USES OF RADIOIODINE

The Department of Radiology of the University of Texas Southwestern Medical School will give an intensive didactic, laboratory and clinical course in the diagnostic and therapeutic uses of radioiodine June 15 through June 20, 1959. The course faculty will be the staff of the Department of Radiology and the Visiting Lecturer will be Leslie J. DeGroot, M.D., Department of Internal Medicine, Harvard University School of Medicine and Massachusetts General Hospital, Boston, Massachusetts.

The course is intended to qualify enrollee for Atomic Energy Commission licensure. Enrollment is limited to ten. Additional information about this course can be obtained by writing Frederick J. Bonte, M.D., Professor and Chairman, Department of Radiology, or John S. Chapman, M.D., Assistant Dean of Postgraduate Education, University of Texas Southwestern Medical School, 5323 Harry Hines Boulevard, Dallas, Texas.

REFRESHER COURSE IN DIAGNOSTIC ROENTGENOLOGY

During the week beginning June 15, 1959, the second annual Refresher Course in Diagnostic Roentgenology will be held at the Cincinnati General Hospital. Tuition is $150.00. Further information and application forms may be obtained from Dr. Benjamin Felson, X-Ray Department, Cincinnati General Hospital, Cincinnati 29, Ohio.

RADIOLOGICAL HEALTH TRAINING COURSES

The U. S. Public Health Service has announced three courses in radiological health training of particular interest to medical personnel and x-ray technicians. There are no tuition, book or other charges for these courses which will be offered at the Robert A. Taft Sanitary Engineering Center, 4676 Columbia Parkway, Cincinnati 26, Ohio.

The courses are:

Medical Aspects of Radiological Health—November 30–December 11, 1959
(Other dates to be announced)
This course is designed primarily for medical, dental and biological personnel of health agencies who have not had radiological health training. The biological effects of radiation, hazard evaluation and protective measures receive major attention. Administrative and legal aspects to the establishment and operation of radiological health programs are also discussed. Acute radiation syndrome and critical organ uptake of radionuclides in animal subjects are studied in the laboratory.

Radiological Health for X-Ray Technicians—July 6–10, 1959
(Other dates to be announced)
This is a basic course for x-ray technicians on the fundamentals of x-ray protection. Lectures cover the atomic structure and radioactivity; properties and production of x-rays; biological effects of radiation; instrumentation; radiation protection and radiological survey procedures. Methods for reducing radiation exposure to the patient are emphasized. Approximately 30 per cent of the time is devoted to laboratory and demonstration sessions.

X-Ray Protection—October 19–23, 1959
This is a more advanced course for health department, industrial and institutional personnel responsible for protection in x-ray installations. Trainees are familiarized with various types of industrial, medical, dental and research x-ray equipment. Detailed instructions are given in procedures and techniques for inspection and survey of x-ray facilities and several laboratory exercises and field inspections of x-ray equipment are conducted.

For application or further information, write to the Center, Att: Chief, Radiological Health Training Activities.

AMERICAN BOARD O RADIOLOGY

The American Board of Radiology will not conduct a Special Examination in Nuclear Medicine (for diplomates in Radiology or Therapeutic Radiology) in June 1959. Candidates desiring this examination will be accommodated on December 5, 1959 at the Shoreham Hotel in Washington, D. C. Deadline for filing applications is August 1, 1959.

The regular fall examination will be held at the Shoreham Hotel, December 6–9, 1959. Deadline for filing applications is July 1, 1959. Candidates examined in Diagnostic Roentgenology may expect to be examined in Physics.

We regret to announce the death on March 18 at Belfast, Maine, of Dr. George W. Holmes, a distinguished member and past President of the American Roentgen Ray Society and also the death on March 28, at Boston, Massachusetts, of Dr. Merrill C. Sosman, a distinguished member and past President of the American Roentgen Ray Society.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

Principles of Radiographic Exposure and Processing. By Arthur W. Fuchs, Rochester, New York. Cloth. Price, $10.50. Pp. 284, with 159 illustrations. Charles C Thomas, Publisher, 301 East Lawrence Avenue, Springfield, Illinois, 1958.

This book should be a standard text for all roentgen-ray technicians and a great help to all radiologists who are not familiar with the advantages of the optimum kilovoltage technique.

At the turn of the century, the author's father produced roentgenograms of excellent diagnostic quality in the Chicago area. With this background, the author began his work in roentgen-ray technique during the first World War; he has spent his lifetime with one of the large roentgen-ray film companies and has actively participated in the teaching program of the United States Army. The book presents, in complete detail, the theory, physical principles, and practical use of roentgen rays in diagnostic medical procedures. Chapters are devoted to the history of the development of roentgen rays, the development of medical roentgen-ray film, the physical principles involved in the exposure of the film, the processing chemistry and many other phases of the subject.

Of especial interest are the chapters on the function of kilovoltage and tube current. The optimum kilovoltage technique developed by the author is completely explained and illustrated with well-chosen roentgenograms. This roentgenographic technique is not to be confused with the so-called high kilovoltage technique, *i.e.*, 100–130 kv.

Your reviewer has had many years of satisfactory experience with Fuchs' optimum kilovoltage technique, but does not wish to convey the idea that this book is just a presentation of a technique chart or outline. It actually is a most complete work making available to the radiologist and technician principles and facts needed for the production of consistently high quality roentgenograms.

The author and publisher are to be complimented on the superior quality of the illustrations and the large, clearly legible type.

Clyde A. Stevenson, M.D.

Elements of Biophysics. By James E. Randall, B.S.E.E., M.S., Ph.D., Associate Professor of Physiology and Biophysics, Department of Physiology, and Pharmacology, University of Missouri Medical Center. Cloth. Price, $8.00. Pp. 333. Year Book Publishers, Inc., 200 East Illinois Street, Chicago, Illinois, 1958.

This text should aid the student of medicine and biology to express many types of physiologic functions more objectively in physical terms of a given vector and scalar quantity. He is also taught how to evaluate such measurements in terms of associated errors and significant statistical variation. The author believes that physical instruments serve to extend the field of biologic measurements and to enhance the ability of the human senses to discriminate biologic changes; however, instrumental limitations should always be properly defined.

In the last several chapters the author examines the physical concepts in mechanics, the kinetic molecular theory, acoustics, fluids, electricity, electromagnetism, atomic and nuclear structure, and radioisotopes relative to defining specific functions of the living organism. A resident in roentgenology should find this volume helpful as a basic review of his training in biophysics.

Jan Nyboer, M.D.

Clinical Radiology of Acute Abdominal Disorders. By Bernard S. Epstein, M.D., Chief, Department of Radiology, The Long Island Jewish Hospital, New Hyde Park, New York; Associate Clinical Professor of Radiology, Albert Einstein College of Medicine, Yeshiva University, New York, New York. Cloth. Price, $15.00. Pp. 352, with 224 illustrations. Lea & Febiger, Washington Square, Philadelphia 6, Pennsylvania, 1958.

The title of this book is a misnomer and the reader who scans through the table of contents will see that the book actually gives a neat and concise presentation of practically every condition of roentgenologic interest of the gastrointestinal tract and associated abdominal organs.

About two or three pages are devoted to each disease process with a clear discussion of the roentgenologic findings. The illustrations are the best the reviewer has ever seen in a book on gastrointestinal roentgenology. The original roentgenograms were of high quality and the author and publisher made good use of localized views to demonstrate the significant changes so that the reader actually gets as much information as if he had the original roentgenograms in his hand.

The limits of roentgenologic examination in the diagnosis of gastrointestinal diseases are emphasized and the need for clinical correlation is adequately discussed. Since each chapter is a somewhat condensed presentation of the material, an adequate list of additional reading is given by the author.

This book will be extremely valuable to the practicing clinical radiologist as well as to any other physician interested in gastrointestinal roentgenology, for, excluding consideration of the esophagus, it presents material which easily could have filled three large volumes.

The book is highly recommended.

Clyde A. Stevenson, M.D.

Lues Connata. By Dozent Dr. Med. Johannes Oehme, Oberarzt der Universitäts-Kinderklinik, Marburg an der Lahn, Früher Leipzig. Second edition. Cloth. Price, DM 26.—. Pp. 116, with 51 illustrations and/34/tables. George Thieme, Hainstrasse 17/19, Aufgang C, Leipzig C 1, Germany, 1957.

This thorough study is based on the author's large experience with congenital syphilis. During the post war years, he treated or followed almost 2,000 children of syphilitic mothers at the pediatric clinic of Leipzig University.

The monograph's first part consists of a critical re-evaluation of the clinical symptoms and signs in congenital syphilis. The author stresses the diagnostic importance of an increased sedimentation rate, the finding of an enlarged liver and the characteristic chronic rhinitis. He calls attention to the fact that hepatosplenomegaly, interstitial pneumonia, rashes and chorioretinitis are nonspecific signs and do occur in other conditions such as erythroblastosis fetalis, toxoplasmosis, Listeria infection and cytomegalic inclusion disease. Of particular interest are the chapters dealing with the roentgenologic findings in congenital lues. The author found that zones of increased (or at times decreased) density near the epiphyseal line and/or double contouring of the tarsal bones are not specific for congenital lues but do occur in cases of maternal toxemia of pregnancy, transfusion reactions, toxoplasmosis and diabetes of the mother. He, therefore, coins for these and similar bone changes the collective term of "enchondral embryopathy." The author's statements on these questions are supported by some experimental and histologic evidence included in the monograph.

The therapeutic measures in congenital syphilis are dealt with at some length. They are divided into: (1) antenatal prophylaxis; (2) postnatal preventive treatment; (3) therapy in the first year of life; and (4) treatment in the preschool and school age.

The author has had excellent results with the prophylactic and preventive use of penicillin, and he recommends it also for the treatment of manifest congenital lues during the first year of life. In cases of congenital syphilis beyond that age, he found that penicillin treatment results in reversal to seronegativity in only 33 per cent within twelve months. Consequently, he recommends for this age group additional courses of salvarsan-bismuth therapy. He does not, however, present evidence for the assumption that this improves the result of therapy. The author favors legislative measures in order to enforce treatment for pregnant women known to be luetic, as is the practice in Eastern Germany where this monograph was published.

Lewis E. Etter, M.D.

X-Ray Diagnosis of the Alimentary Tract in Infants and Children. By Edward B. Singleton, M.D., Director of Radiology, Texas Children's and St. Luke's Hospitals, Houston, Texas; Clinical Assistant Professor of Radiology, Baylor University College of Medicine; Clinical Associate Radiologist, University of Texas Post Graduate School. Cloth. Price, $11.00. Pp. 352, with numerous illustrations. The Year Book Publishers, Inc., 200 East Illinois Street, Chicago, Illinois, 1959.

This excellent volume provides a thorough review of the clinical and roentgenologic aspects of all of the entities coming to the attention of the radiologist in pediatric gastrointestinal work.

Following introductory chapters on indications for and techniques of examination, there is a sound logical arrangement of chapters on the normal roentgenologic appearance, the roentgenologic manifestations of congenital abnormalities, and the changes in acquired abnormalities of each of the subdivisions of the gastrointestinal tract. Chapters other than these are one on the abnormalities of the gastroesophageal junction, interposed in the section on the esophagus, and a final chapter on congenital diaphragmatic hernia. Excellent bibliographies appended to each chapter indicate the pertinent literature.

The emphasis on radiation protection is excellent, as is the discussion of the always fascinating lower esophagus. The embryologic correlations are well done as background for understanding of congenital anomalies. The limitations of the roentgen-ray findings are accurately delineated with good emphasis on those situations in which further roentgen examination should be abandoned in favor of immediate laparotomy.

The index is complete and usable, with the practical feature of asterisks to indicate those topics covered by illustrations. An interesting cover shows the roentgen appearance of a partially opacified gastrointestinal tract superimposed on a photograph of a statue of a child.

This concise yet complete and accurate summary of pediatric gastrointestinal roentgen findings deserves a place on the desk of pediatricians and radiologists alike.

Walter M. Whitehouse, M.D.

The Scandinavian Journal of Clinical & Laboratory Investigation. Volume 10—Supplementum 33. The Synthesis of Prothrombin and Proconvertin in the Reticulo-Endothelial System. An experimental study on rats. By Pär Slätis. Paper. Pp. 83. Helsingfors, OyWeilin & Göös Ab, 1958.

This monograph demonstrates that the reticuloendothelial system (both within and without the liver) plays a major role in the synthesis of prothrombin and proconvertin in rats. The method used involved blockading the reticuloendothelial system by the use of thorotrast and demonstrating a rapid fall in the blood levels of prothrombin and proconvertin. In addition, it was shown that in normal rats the reduction in these substances following the action of dicumarol could be reversed rapidly by vitamin K_1, but in blockaded rats this could not be accomplished, suggesting that vitamin K_1 also acts in the reticuloendothelial system. As dicumarol has been shown to exert its effect in the liver, this leaves unexplained the corrective action of vitamin K_1 (acting in the reticuloendothelial system) on dicumarolized rats. Additional evidence of an extrahepatic source for prothrombin and proconvertin is the fact that administration of vitamin K_1 to hepatectomized rats prevented the precipitous fall in the levels of these substances which otherwise occurred.

A. R. W. Climie, M.D.

Radiation Protection. By Carl B. Braestrup, Director, Physics Laboratory, Frances Delafield Hospital; Associate, Department of Radiology, Columbia University, New York, New York; and Member, Executive Committee, National Committee on Radiation Protection and Measurements; and Harold O. Wyckoff, Chief, Radiation Physics Laboratory, National Bureau of Standards, Washington, D. C.; and Secretary, International Commission on Radiological Units and Measurements. Cloth. Price, $10.50. Pp. 361, with figures and tables. Charles C Thomas, Publisher, 301 East Lawrence Avenue, Springfield, Illinois, 1958.

Prior to the advent of nuclear energy, radiation protection interested only those in radiology and a small number of industrial users of the roentgen ray and radium. The tremendous increase in the use of radiation, coupled with the newer knowledge of low dosage radiation effects, has focused the attention of many persons outside of radiology on this field. This highly technical subject has become of interest to public health officials, industrial hygienists, safety engineers and other individuals outside of the purview of radiology and outside of medicine. It is presumably for this large and varied group that this book was written.

The authors have presented an authoritative and comprehensive survey of the science of radiation protection in this volume. The first chapter is a short historical introduction to the radiation protection problem. This is followed by a chapter on the production and attenuation of radiation. Unfortunately, the authors have presumed some background in physics for understanding this chapter and it may well discourage those readers who are frightened by the sight of physical mathematical formulae. The following chapter concerns the measure-

ment of radiation and is equally as technical and may also discourage some readers. Both of these chapters are comprehensive and have excellent bibliographies.

A general discussion of shielding methods and materials is presented by the authors in a subsequent chapter which also depicts the general approach to the use of protective methods and materials.

The specific problems of medical roentgen ray installations are covered in detail as are the special problems of dental roentgen ray installations. The protection problems of industrial and other nonmedical installations are described in a separate chapter as are megavoltage installations.

Radiation emitting materials are discussed comprehensively in two chapters, one on sealed radioactive sources and another on nonsealed radioactive sources.

Dr. Merrill Eisenbud of the United States Atomic Energy Commission has written a detailed chapter on atmospheric contamination by radioactive substances. The biologic effects of radiation are quite adequately covered by Drs. Titus C. Evans, and S. Allan Lough of the College of Medicine, University of Iowa and the Atomic Energy Commission, respectively.

The appendix contains a great deal of useful data for those working in or concerned with this field.

The book is recommended highly as an authoritative, comprehensive treatise on the subject for those already having some background in this area. The reviewer, however, is not in accord with the publisher in his statement on the flyleaf that the volume may be useful to dermatologists, dentists, and similar medical users of radiation. For such users, it is this reviewer's belief, a simple introductory text on the subject yet remains to be written.

George Tievsky, M.D.

BOOKS RECEIVED

Modern Trends in Diseases of the Vertebral Column. Edited by Reginald Nassim, B.M., F.R.C.P., Physician to St. George's Hospital, Physician to the Royal National Orthopaedic Hospital, London; and H. Jackson Burrows, M.D., F.R.C.S., F.R.A.C.S., Orthopaedic Surgeon to St. Bartholomew's Hospital, and to the Royal Institute of Orthopaedics, British Postgraduate Medical Federation (University of London), Consulting Orthopaedic Surgeon to the Royal Navy. Cloth. Price, $15.00. Pp. 292, with 172 illustrations. Paul B. Hoeber, Inc., 49 East 33rd Street, New York 16, New York, 1959.

Cancer: Diagnosis and Treatment. Edited by John B. Field, M.D., Ph.D., Assistant Clinical Professor of Medicine, University of Southern California, School of Medicine, Los Angeles, California. With 28 contributors. Cloth. Price, $18.50. Pp. 796, with numerous illustrations. Little, Brown & Company, 34 Beacon St., Boston 6, Massachusetts, 1959.

Fracture Surgery. A Textbook of Common Fractures. By Henry Milch, M.D., Emeritus Attending and Consulting Orthopedic Surgeon, Hospital for Joint Diseases, New York, and Robert Austin Milch, M.D., Assistant Resident Surgeon, Peter Bent Brigham Hospital, Boston, with a chapter on anesthesia by Herbert D. Dubovsky, M.D., Director of Anesthesiology, Easton Hospital, Easton, Pennsylvania. Cloth. Pp. 470 with 671 illustrations. Price, $17.50. Paul B. Hoeber, Inc., 49 East 33rd Street, New York 16, New York, 1959.

Regeneration in Vertebrates. Edited by Charles S. Thornton. Cloth. Price, $3.25. Pp. 108, with numerous illustrations. The University of Chicago Press, 5750 Ellis Avenue, Chicago 37, Illinois, 1959.

Cytodifferentiation. Edited by Dorothea Rudnick. Cloth. Price, $3.75. Pp. 148, with 26 illustrations. The University of Chicago Press, 5750 Ellis Avenue, Chicago 37, Illinois, 1958.

Environmental Influences on Prenatal Development. Edited by Beatrice Mintz. Cloth. Price, $3.00. Pp. 87, with 26 illustrations. The University of Chicago Press, 5750 Ellis Avenue, Chicago 37, Illinois, 1958.

Atomic Medicine. Edited by Charles F. Behrens, M.D., F.A.C.R., Rear Admiral, MC, U. S. Navy, (ret.); Roentgenologist, Yater Clinic, Washington, D. C.; Consultant and Lecturer in Radiology, U. S. Naval Medical Center, Bethesda, Maryland; Formerly: Director, Atomic Defense Division, Bureau of Medicine and Surgery, Navy Department; Commanding Officer, Navy Medical Research Institute, National Naval Medical Center, Bethesda, Maryland. Third Edition. Cloth. Price, $15.00. Pp. 705, with numerous illustrations. The Williams & Wilkins Company, Mount Royal and Guilford Avenues, Baltimore 2, Maryland, 1959.

A Guide to Nuclear Energy. By R. F. K. Belchem, C.B., C.B.E., D.S.O., American Machine & Foundary Company; Formerly with T. I. Nuclear Engineering, Ltd. Cloth. Price, $3.75. Pp. 77, with 13 illustrations. Philosophical Library, Inc., 15 East 40th Street, New York 16, New York, 1958.

Radioactivity Measuring Instruments: A Guide to Their Construction and Use. M. C. Nokes, Atomic Energy Research Establishment, Harwell. Cloth. Price, $4.75. Pp. 75, with 38 illustrations. Philosophical Library, Inc., 15 East 40th Street, New York 16, New York, 1958.

Radiographic Atlas of Skeletal Development of the Hand and Wrist. Second Edition. By William Walter Greulich, Professor Anatomy, Stanford University School of Medicine, and S. Idell Pyle, Research Associate, Department of Anatomy, Western Reserve University and Stanford University Schools of Medicine. Cloth. Price, $15.00. Pp. 256, with numerous illustrations. Stanford University Press, Stanford, California, Oxford University Press, London, 1959.

The Physiopathology of Cancer. By 32 authors. Second Edition. Edited by Freddy Homburger, M.D.; Foreword by Joseph C. Aub, M.D., Sc.D. Cloth. Price, $33.00. Pp. 1180, with 189 illustrations. Paul B. Hoeber, Inc., 49 East 33rd Street, New York 16, New York, 1959.

Therapeutic Radiology: Rationale, Technique, Results. By William T. Moss, M.D., Assistant Professor of Radiology, Northwestern University School of Medicine, Department of Radiology, Chicago, Illinois; Director, Department of Therapeutic Radiology, Chicago Wesley Memorial Hospital; Chief, Department of Therapeutic Radiology, Veterans Administration Research Hospital, Chicago, Illinois. With a Foreword by Lauren V. Ackerman, M.D. Cloth. Price, $12.50. Pp. 403, with 146 illustrations. The C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, Missouri, 1959.

Acta Orthopaedica Scandinavica. Supplementum No. 36. Tensile Strength of the Whole-Thickness Skin Graft Used as Replacement of Tendon and Ligament Defects. An Experimental Study. By Tuula Jokinen. Paper. Pp. 91, with 16 illustrations and numerous tables. Ejnar Munksgaards Forlag, Nørregade 6, Copenhagen, Denmark, 1958.

The Scandinavian Journal of Clinical & Laboratory Investigation. Vol. 10—Supplementum 36. Calcium Requirement and Adaptation in Adult Men. By Ole J. Malm. Paper. Pp. 290, with numerous tables. Oslo University Press, Oslo, Norway, 1958.

Symposium on Genetic Approaches to Somatic Cell Variation. Given at Research Conference for Biology and Medicine of the Atomic Energy Commission. Sponsored by The Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee. Held at Gatlinburg, Tennessee, April 2–5, 1958. Paper. Pp. 410, with numerous illustrations. The Wistar Institute of Anatomy and Biology, Philadelphia, Pennsylvania, 1958.

Atlas of Tumor Pathology. Section XII—Fascicle 40. Transplantable and Transmissible Tumors of Animals. By Harold L. Stewart, M.D., Katharine C. Snell, M.D., Lucia J. Dunham, M.D., and Samuel M. Schlyen, M.D., Laboratory of Pathology, National Cancer Institute, National Institutes of Health, Bethesda, Maryland. Paper. Price, $3.50. Pp. 378, with 287 illustrations. Armed Forces Institute of Pathology, under the auspices of the Subcommittee on Oncology, of the Committee on Pathology, of the Division of Medical Sciences, of the National Academy of Sciences—National Research Council, Washington, D. C., 1959. For sale by the American Registry of Pathology, Armed Forces Institute of Pathology, Washington 25, D. C.

L'oesophage. By Guy Albot, Médecin de l'Hôtel-Dieu; and Felix Poilleux, Chirurgien de l'hôpital Corentin-Celton. Paper. Price, 3,600 fr. Pp. 274, with 112 illustrations. Masson et Cie, Éditeurs, 120 Boulevard Saint-Germain, Paris 6, France, 1958.

Acta Radiologica Supplementum 166. Radioactive Sulphur As Benzidine Sulphate. A Methodological Study with Special Reference to S^{35}-Labelled Penicillin in Oral Surgery. By Erik Ågren. Paper. Price, Sw.Kr. 30:–. Pp. 108, with 32 illustrations and 23 tables. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1958.

Acta Radiologica Supplementum 167. Time-Dose Relationship and Morphology of Delayed Radiation Lesions of the Brain in Rabbits. By Nils O. Berg, and Martin Lindgren. Paper. Price, Sw.Kr. 30:–. Pp. 118, with 72 illustrations and 19 tables. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1958.

Acta Radiologica Supplementum 168. The Radiation Reaction in the Vaginal Smear and Its Prognostic Significance. Studies on Radiologically Treated Cases of Cancer of the Uterine Cervix. By Olle Kjellgren. Paper. Price, Sw.Kr. 35:–. Pp. 170, with 26 illustrations and 31 tables. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1958.

Acta Radiologica Supplementum 169. Late Results of Radium Therapy in Cervical Carcinoma. A Clinical-Statistical Study on 789 Patients Treated at The Radium Centre, Copenhagen, during the Period 1922–1929. By Bent Sørensen. Paper. Price, Sw.Kr. 25–. Pp. 190, with 44 illustrations and 41 tables. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1958.

Acta Radiologica Supplementum 170. On Tolerance of Brain Tissue and Sensitivity of Brain Tumors to Irradiation. By Martin Lindgren. Paper. Price, Sw.Kr. 25:–. Pp. 73, with 30 illustrations and 8 tables. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1958.

Acta Radiologica Supplementum 172. Aortic Valvular Diseases Studied by Percutaneous Thoracic Aortography. By Per Ödman and Jan Philipson. Paper. Price. Sw.Kr. 25:–. Pp. 59, with 21 illustrations. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1958.

VOL. 81, NO. 5

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. LEUCUTIA, M.D., Harper Hospital, Detroit 1, Michigan

INDEX TO ABSTRACTS

ROENTGEN DIAGNOSIS

HEAD

HERNÁNDEZ, A.: New radiographic technique for projection of the anterior ethmoid cells; (technique of Esteban Hernández) 907
AGNOS, J. W., and WOLLIN, D. G.: The effect of rotation of the skull on the measured position of the pineal gland 907
BALOGH, K., and BALOGH, K., JR.: Symmetrical fibrous swelling of the mandible with changes in the dental pulp 907
CHAVANY, J.-A., and METZGER, J.: Abnormal or misleading clinical forms of osteomas of the cranial vault 907
CHIAT, H., and KITTREDGE, R. D.: Cholesteatoma of the temporal bone 908
JANZEN, R., and DIECKMANN, H.: Neurological symptoms due to skeletal malformations of the cervico-occipital region 908
WHOLEY, M. H., BRUWER, A. J., and BAKER, H. L., JR.: The lateral roentgenogram of the neck. (With comments on the atlanto-odontoid-basion relationship) 908
ELKINGTON, J. St. C.: Cerebral vascular disease in the light of modern techniques 909
CRAWFORD, E. S., DEBAKEY, M. E., and FIELDS, W. S.: Roentgenographic diagnosis and surgical treatment of basilar artery insufficiency 909
GHIRARDI, L., GARELLO, L., and LERTORA, M.: Experience with the submento-bregmatic projection in cerebral angiography 910

NECK AND CHEST

MURRAY, J. P.: Neuromuscular and functional disorders of the pharynx 910
MADORE, P., and GLAY, A.: Oesophagobronchial fistula complicating oesophageal diverticulum 911
WATKINS, D. H.: Surgical complications in the thorax of staphylococcal pneumonitis 911
PANTONE, A. M.: Radiographic chest examination of the tuberculous patient 912
JACOBS, L. G.: Pulmonary torulosis 912
MELAMED, M.: Roentgen evaluation of the chest after thoracic surgery 912
ODERR, C. P., PIZZOLATO, P., and ZISKIND, J.: Emphysema studied by microradiology 913
LÜBKER, H.: A contribution to the laminagraphic technique of the bronchial tree 913
BONTE, F. J., and REYNOLDS, J.: Hydrocarbon pneumonitis 913
NELSON, S. W., and CHRISTOFORIDIS, A.: Reversible bronchiectasis 914
PFUETZE, K. H., and PYLE, M. M.: Medical management of pulmonary tuberculosis 914
LANGSTON, H. T.: The surgery of pulmonary tuberculosis 914
ROTH, M.: Fluid absorption from the lung, as regards bronchographic contrast substances 915
ROWE, C. W.: Increasing scope of bronchography with dionosil 915
LODWICK, G. S., KEATS, T. E., and DORST, J. P.: An evaluation of the significance of transverse hilar measurements in the diagnosis of primary lung cancer 916
PUNCHETTI, V., IONESCU, L., and CUBILLOS, L.: Gastroenterogenic mediastinal cysts 916
STEINBERG, I., and SAMMONS, B. P.: Aneurysmal dilatation of the aortic sinuses in coarctation of the aorta: Report of two new cases and review of the literature 916
HOLMAN, C. W., and STEINBERG, I.: The role of angiocardiography in the surgical treatment of massive pericardial effusions 917
LULL, G. F., JR.: Pericardial celomic cyst; a reevaluation 917
CIARPAGLINI, L., and IANNACCONE, G.: Dynamic changes of the varicose esophagus; cineroentgenographic examination with the image amplifier 917
BALOW, R. M., and WIETERSEN, F. K.: Postemetic rupture of the esophagus; a report of three cases 918

ABDOMEN

GLENDINNING, A. C.: Barium meal and follow-through 918
ELLIS, K.: Gastrojejunal ulcer 918
STEIN, J., PERLMAN, B. B., and POVALSKI, A.: Gastroduodenal intussusception 919
SOTEROPOULOS, C., and GILMORE, J. H.: Roentgen diagnosis of acute appendicitis 919
DUBRA, C. C.: Encysted choleperitoneum 920
GRAY, E. D.: Some radiological aspects of biliary tract disease. President's address 920

Milanés Alvarez, F., Conde Otero, F., Rodrígues Remus, C., Eiber Haft, E., Trujillo Ibarra, C., and Hernández Machado, J.: Value of intravenous biligrafin in the diagnosis of post-cholecystectomy syndrome 920
Boni, R.: Triple gallbladder and calcareous bile 920

Gynecology and Obstetrics

Tetti, A., and Barbanti, A.: The diagnostic possibilities of amniography 921
Breen, J. L., and Riva, H. L.: Polyostotic fibrous dysplasia and pregnancy; a case report 921
Maurer, H.-J., and Wendt, H.: The relationship of the incidence of carcinoma to the month-of-birth and to blood group 921
Woltz, J. H. E., Bradford, W. Z., Bradford, W. B., and McCoy, J. B., Jr.: Complications of hysterosalpingography 921

Genitourinary System

Herman, J. R., Mason, B., and Light, I.: Fluorine in urinary tract calculi 922
Marquardt, C. R., Pick, J. W., Melamed, A., Marck, A., and Holt, A. H.: The use of miniature film (renogram) for kidney stone surgery 922
Dubilier, W., Jr., and Evans, J. A.: Peripelvic cysts of the kidney 922
Ross, J. A.: Peri-ureteritis fibrosa, with notes on three cases 923
Peart, W. S., and Sutton, D.: Renal-vein catheterisation and venography; a new technique 923
Sproul, R. D., Fraser, R. G., and MacKinnon, K. J.: Aneurysm of the renal artery .. 924
Spring, M., and Gross, S. W.: Ruptured aneurysm of the circle of Willis associated with polycystic kidneys 924
Whitley, J. E.: A simple protection device for urologic units 924

Nervous System

Psenner, L., and Schönbauer, E.: On tuberous sclerosis with special consideration of roentgenologic symptomatology 924

Skeletal System

Calandi, C., and Vichi, G. F.: Clinical and radiologic study of a case of gargoylism (Pfaundler-Hurler disease) 925
Broder, H. M.: Prognosis in Legg-Perthes disease 925
Stanisavljevic, S., and St. John, E. G.: Congenital fusion of three lumbar vertebral bodies 926
Rezek, J.: On arthrography 926
Osserman, E. F.: Natural history of multiple myeloma before radiological evidence of disease. Memorial Fund Lecture 926

Blood and Lymph System

Gerard, F. P., and Lyons, H. A.: Anomalous artery in intralobar bronchopulmonary sequestration; report of two cases demonstrated by angiography 927
Rösch, J., Bret, J., and Lišková, M.: Splenoportography in the diagnosis of splenomegaly 927

MISCELLANEOUS

Gerstner, H. B.: Acute clinical effects of penetrating nuclear radiation 928

ROENTGEN DIAGNOSIS

Head

Hernández, Antonio. Nueva técnica radiografica para la projección de las celdas etmoidales anteriores; (técnica de Esteban Hernández). (New radiographic technique for projection of the anterior ethmoid cells [Technique of Esteban Hernández].) *Acta ibér. radiol. cancerol.*, Oct.–Dec., 1957, *13*, 471–490.

In the examination of the paranasal sinuses, three routine exposures are made on a single 18×24 film: one in a mentonasal position, for visualization of the maxillary and frontal sinuses; and one each in the right and left views of the anterior ethmoid cells, using a new technique. For this new technique the patient is placed in a prone position with the mouth closed and the nose and chin supported on a cassette; the head is rotated 8° from the perpendicular, first to the right then to the left. The central beam passes above the base of the nose. The head is immobilized by an auxiliary brace.

The roentgenograms obtained with this technique show the ethmoid cells and their extensions. Positioning of the patient is easy and can be done rapidly. There is also an economy in the use of a single film.—*Murray M. Friedman, M.D.*

Agnos, John W., and Wollin, D. G. The effect of rotation of the skull on the measured position of the pineal gland. *J. Canad. A. Radiologists*, Sept., 1958, *9*, 40–44. (From: The Department of Diagnostic Radiology, University of Toronto, Faculty of Medicine, and the Toronto General Hospital, Toronto, Ontario, Canada.)

Every radiologist has misgivings in assessing the position of the pineal gland in roentgenograms showing rotation. For reasons given by Fray, the pineal displacement measuring up to 2 mm. from the midline was considered normal.

The authors carried out similar experiments by using symmetric and asymmetric skulls of dolichocephalic, mesocephalic, and brachycephalic shape. Employing a 50 inch target-film distance, roentgenograms were made in the Towne, Caldwell and reverse Caldwell views, with 0, 5, 10, 15 and sometimes 20 degrees of rotation both to the right and to the left. Lateral roentgenograms were also made with similar degrees of rotation (chin towards film).

In skulls of all three shapes, whether symmetric or asymmetric, the degree of permissible rotation varied inversely as the cephalic index of the skull and decreased markedly from Caldwell to either Towne or reverse Caldwell projections. In the lateral projection a rotation of 20° produced no significant changes in the measured position of the pineal gland.—*J. T. Ling, M.D.*

Balogh, K., and Balogh, K., Jr. Symmetrical fibrous swelling of the mandible with changes in the dental pulp. *Oral Surg., Oral Med., & Oral Path.*, Nov., 1958, *11*, 1261–1268. (From: School of Medicine and Dentistry, Budapest University, Budapest, Hungary.)

This is a report of a case of familial fibrous swelling of the jaws (cherubism). The patient was an eight year old boy with progressive painless enlargement of the mandible for one year. Roentgenograms revealed multilocular cyst-like mandibular lesions. Bilateral curettage was carried out. Satisfactory bone regeneration was evident fourteen months later, and there had been no progression of the lesions. The authors believe that surgical intervention is indicated in active bilateral cases.

The patient's father had a similar mandibular configuration, but roentgenograms revealed no lesions. However, some authors believe that in adult life, the multilocular rarefactions are poorly defined or absent, and that the entire mandible may appear sclerotic.

A total of 15 affected families have been reported so far. Only 4 of these families were observed outside the U.S.A.: 1 in France, 1 in South America, and 2 in England.—*Eugene A. Cornelius, M.D.*

Chavany, J.-A., and Metzger, J. Formes cliniques anormales ou trompeuses des ostéomes de la voute du crane. (Abnormal or misleading clinical forms of osteomas of the cranial vault.) *Presse méd.*, Oct. 29, 1958, *66*, 1677–1680. (From: Clinique neuro-chirurgicale et Laboratoire de neuro-radiologie de l'Hôpital de la Pitié, Paris, France.)

The neurologic and general physical examinations of cases of skull osteomas are usually negative. Only exceptionally are there findings, and this happens when there is growth toward the interior of the skull. The diagnosis is ordinarily made by roentgenographic examination. Another rule is that occipital osteomas are rare (8 per cent) while frontal or fronto-ethmoidal tumors are common (70 per cent). Five cases are reported which for various reasons are exceptions to the rule. The first three entered the hospital on account of pain. The fourth presented no apparent mass. The fifth was considered in error as the principal diagnosis when the clinical course revealed a deep-seated tumor with no relation to the bony neoplasm.

The roentgenographic examination requires, in addition to the standard views, tangential views made with different densities, magnification techniques using an 0.3 mm. focal spot and laminagraphy. The osteoma presents as an osseous prolifera-

tion without clear structure and a density greater than that of the normal vault. Its density is relatively homogeneous, diminishing from the center toward the edges. The lesion is outside of the normal course of the outer table and is continuous with it or slightly separated. At the greatest prominence the diploe appears somewhat thickened, condensed and with a partial loss of its trabecular structure. Neighboring vascularization is normal.

The differential diagnosis as well as the significance of the hyperostotic process in relation to meningioma are discussed.—*Marcy L. Sussman, M.D.*

Chiat, Harold, and Kittredge, Richard D. Cholesteatoma of the temporal bone. *Radiology*, Oct., 1958, *71*, 559–562. (Address: H. Chiat, 525 68th St., New York 21, N. Y.)

A review of 25 proved cases of cholesteatoma of the temporal bone is presented by the authors. In 13 instances a correct preoperative roentgen diagnosis had been made. Of the projections used in the routine examination of the mastoids the fronto-occipital projection will most often and most clearly demonstrate the defect characteristic of a cholesteatoma. Laminagraphic studies often will clearly demonstrate the destructive changes when they can only be suspected on the routine roentgenograms, thus increasing the chances of making a correct preoperative diagnosis.—*Donald N. Dysart, M.D.*

Janzen, R., and Dieckmann, H. Neurological symptoms due to skeletal malformations of the cervico-occipital region. *German med. Monthly*, Nov., 1958, *3*, 335–338; original article published in the *Deutsche med. Wchnschr.*, 1958, *83*, 1077. (From: The Neurological Department, Dortmund Municipal Hospitals, Dortmund, Germany.)

Cervico-occipital skeletal malformations and the neurologic symptoms which sometimes accompany them are discussed. Those which can be roentgenologically diagnosed are: (1) basilar impression or platybasia; (2) fusion of the atlas with the occipital bone; (3) a large dens of the axis; and (4) aplasia of the basilar portion of the occipital bone. By far the most common is platybasia.

The skeletal malformations, particularly platybasia, often occur without neurologic abnormalities, but, when present, a syndrome similar to that seen with a posterior fossa or upper cervical tumor is produced. Onset of symptoms is usually gradual with paresthesia, headache or vertigo. Pressure or traction on the fifth through the twelfth cranial nerves occurs and upper cervical nerve lesions produce paralysis in the shoulder girdle region. The findings are easily confused with progressive muscular dystrophy or amyotrophic lateral sclerosis. Aside from localizing signs, the appearance of the patient is often characteristic, *i.e.*, short neck, head deviated to one side with limited motion or an elevated shoulder.

This constellation of findings makes roentgenologic examination mandatory. Survey roentgenograms should be supplemented by laminagraphy. If the atlanto-occipital joint is not clearly seen on the "open-mouth" view of the upper cervical spine, malformation in this zone should be suspected. Basilar impression is diagnosed using the intermastoid line and Chamberlain's line as criteria.

The authors collected 42 cases in two and one-half years, which is evidence that the condition is not rare. Brief summaries of 8 cases are presented.—*Jack Reynolds, M.D.*

Wholey, Mark H., Bruwer, André J., and Baker Hillier L., Jr. The lateral roentgenogram of the neck. (With comments on the atlanto-odontoid-basion relationship.) *Radiology*, Sept., 1958, *71*, 350–356. (Address: M. H. Wholey, The Mayo Clinic, Rochester, Minn.)

The authors have reviewed some 600 normal lateral cervical roentgenograms at the Mayo Clinic in order to establish certain normal measurements to serve as a useful guide in evaluating the bone and soft tissue relationships of the neck. These roentgenograms were made with the patient sitting erect and with the neck in the neutral position. The measurements of the retropharyngeal space, retrotracheal space, and the anteroposterior diameter of the spinal canal were tabulated and an average and normal range figure established. The authors feel that sagittal measurements of the retropharyngeal space greater than 14 mm. in children and 22 mm. in adults warrant further investigation to rule out pathologic entities. Any localized irregularity of the posterior tracheal wall was regarded as significant until proved otherwise as hypertrophic ridging was not found to influence this area as it does the barium filled esophagus. Such localized bulging of the posterior tracheal wall may be due to neoplasm, hematoma, or inflammation. The presence of a bolus of air in the cervical esophagus was not regarded as significant in most cases.

The authors feel that the sagittal diameter of the spinal canal is established early in life as the range for children corresponded to that of adults. These measurements ranged from 22 mm. at the first cervical vertebra to 18 mm. at the seventh. An increase in the sagittal diameter is perhaps more indicative of abnormality than is a decreased measurement as an extramedullary tumor, such as a neurofibroma, may expand enough to cause an abnormal diameter.

The atlanto-odontoid-basion relationship is discussed and it is felt that this relationship is of more practical significance than either Chamberlain's or McGregor's line. The middle half of the upper end of the odontoid process normally lies di-

rectly beneath the basion. and on an average of 5 mm. from it. This measurement may normally be increased in infants and small children. The anterior surface of the odontoid is usually within 1 or 2 mm. of the anterior arch of the atlas. Alterations of the atlanto-odontoid-basion relationship are seen in traumatic subluxations of the odontoid with disruption of the transverse ligament of the atlas, in rheumatoid spondylitis and rheumatoid arthritis, and in congenital anomalies.

The authors also discuss and define platybasia and basilar impression.—*John S. Alexander, M.D.*

Elkington, J. St. C. Cerebral vascular disease in the light of modern techniques. *Lancet*, Aug. 2, 1958, *16*, 327–333. (From: St. Thomas's Hospital, London, S.E.1; and The National Hospital, Queen Square, London, W.C.1, England.)

The discussion of subarachnoid hemorrhage is limited to "spontaneous" hemorrhage to differentiate those cases where the bleeding into the subarachnoid space is the primary event from those in which it is secondary. Before arteriography was available, workers depended for diagnosis either on clinical judgement or on necropsy findings. The former introduces many possibilities of error, while the latter inevitably gives a distorted picture since only the cases with fatal outcome can be examined. Also, the analysis of the relatively selected cases in a surgical clinic is likely to give results that are not strictly comparable to those obtained from a general hospital where cases are unselected. Therefore, it is difficult to estimate the relative frequency of the possible causes of subarachnoid hemorrhage; but the more searching the investigation, the greater is the importance attached to the rupture of some pre-existing vascular anomaly of which "berry" aneurysms constitute the great majority and angiomatous malformations a small minority.

The author then details a review of the literature, principally articles by McKissock and Walsh and reaches the following conclusions: (1) subarachnoid hemorrhage treated conservatively carries a grievous mortality; (2) active treatment can be logically applied only after a diagnosis has been made by bilateral carotid angiography which should be carried out as soon as possible; (3) those cases within eight weeks after hemorrhage are, in general, best treated by surgical means; (4) if more than eight weeks have elapsed since the last hemorrhage, conservative treatment is usually preferred to surgery. The question of whether surgical treatment is indicated during the phase of acute hemorrhage remains for the future to decide. In none of the series cited was artificial hyperthermia generally employed.

Intracranial hemorrhage, or localized hemorrhage within the substance of the brain, referred to as an intracerebral hematoma, occurs under two widely different circumstances: (a) by rupture of some preexisting localized vascular anomaly, or (b) by rupture of an arteriosclerotic vessel. A rupture of a cerebral aneurysm usually gives rise to a subarachnoid hemorrhage but may produce a space occupying lesion within the brain, or the two may be associated. The clinical picture is confirmed by the shift of the cerebral arteries revealed by angiography. It is generally agreed that the presence of a massive intracerebral clot in cases of subarachnoid hemorrhage is further indication for surgical intervention.

The diagnosis of an intracerebral hematoma resulting from angiomatous malformations depends upon the clinical findings of a rapidly developing, expanding, intracranial lesion in an otherwise healthy person and the diagnosis is confirmed by angiography. In addition to demonstrating evidence of displacement of the cerebral vessels, the characteristic features of the causative lesions are also demonstrated. Such hematomas should be treated surgically, at the earliest opportunity, by evacuation of the clots and excision of the malformation if this is possible.

Intracranial hematomas resulting from cerebral vascular degeneration with hypertension represent a common and usually terminal event. Some patients live on with varying degrees of disability and they may survive for a long time. If an intracerebral hematoma is suspected, its presence can often be confirmed by demonstrating the shift of the pineal shadow on a roentgenographic examination of the skull. Air ventriculography may be needed to demonstrate the presence of an expanding intracranial lesion.

Three cases are reported and the author concludes that surgical treatment has an undoubted part to play both as a life saving measure and as a means of minimizing the residual disability in certain selective cases.—*John F. Weigen, M.D.*

Crawford, E. Stanley, DeBakey, Michael E., and Fields, William S. Roentgenographic diagnosis and surgical treatment of basilar artery insufficiency. *J.A.M.A.*, Oct. 4, 1958, *168*, 509–514. (From: The Cora and Webb Mading Department of Surgery and the Department of Neurology, Baylor University College of Medicine, and the Methodist Hospital, Houston, Texas.)

Atheromatous obstructions impairing the cerebral circulation may involve the vertebral as well as the carotid arteries. Frequently, the occlusive process is in the neck or thorax with the artery being relatively normal proximal and distal to the lesion and certain operative procedures may restore the normal circulation. The localized lesion may be removed by endarterectomy, or in case of a more extensive lesion the occlusion may be bypassed by a graft. The authors have applied this technique in 43 lesions

occurring in the internal carotid, innominate, subclavian, and left common carotid arteries. Circulation was restored in all patients with relief of the neurologic deficits which occurred prior to surgery.

Atherosclerotic lesions may also occur in the basilar arterial system. Recent studies in patients dying of cerebrovascular accidents show that 39 per cent had basilar artery insufficiency from obstructive lesions frequently located in the cervical segments of the vertebral arteries. Simultaneous occlusion of the internal carotid and vertebral arteries was found in 30 per cent of the cases. This is important because the basilar artery through the circle of Willis is an important source of collateral circulation in patients with carotid occlusion. Because of the frequency with which the two main routes are simultaneously involved, it is important to have a therapeutic attack designed to restore blood flow in the posterior cerebral circulation.

The authors state that the present report is to indicate one's ability to accurately diagnose and surgically treat lesions located primarily in the vertebral artery producing basilar artery insufficiency. A case of a fifty-six year old man who suddenly developed total blindness, paralysis of the right side, partial paralysis of the left side of his body, loss of speech, and feeling of numbness over his entire body is reviewed. These symptoms gradually became less severe. Bilateral carotid arteriograms were normal. The bilateral nature of the patient's symptoms which began with total blindness suggested basilar artery insufficiency and indicated necessity of visualizing the entire cervical segments of the vertebral arteries. The usual method of vertebral arteriography was believed to be inadequate because the proximal portions are not visualized; therefore, a method of injecting the subclavian arteries with 20 cc. of 50 per cent hypaque was considered, whereby the entire vertebral arteries are demonstrated. This procedure was carried out on the patient and showed complete occlusion of the right vertebral artery and partial occlusion of the left vertebral artery at its origin from the subclavian. By a combined cervical thoracic incision, splitting the sternum in the midline, the origin of the left vertebral artery was located and the authors describe their method of resecting an atheromatous plaque from the hypertrophied intima. The patient made a satisfactory recovery.

The authors feel that in this case there was a complete occlusion of the right vertebral artery by a thrombus distal to an atheromatous lesion and that the same process would have occurred on the left side if the atheromatous lesion in the left vertebral artery had not been removed. This was necessary because total basilar insufficiency would not be consistent with survival. Their operation was to (1) remove the obstruction in one vertebral artery and thereby restore normal basilar artery circulation and (2) prevent progression of the disease to complete occlusion by superimposed thrombosis.

The clinical significance of this case lies in the demonstration by roentgenographic examination of a localized lesion in the vertebral artery causing basilar artery insufficiency and in removal of this lesion by surgery. The importance of the fact that many occlusive lesions are extracranial and may be corrected by direct surgical attack is stressed.—*E. F. Koch, Jr., M.D.*

Ghirardi, L., Garello, L., and Lertora, M. Esperienze angiografiche cerebrali in proiezione sottomento-bregmatica. (Experience with the submento-bregmatic projection in cerebral angiography.) *Ann. radiol. diag.*, 1958, *31*, No. 2, 102–118. (From: Clinica delle Malattie Nervose e Mentali, and Istituto di Radiologia, Università di Genova, Genova, Italy.)

The authors have added the submento-bregmatic view to the routine frontal and lateral views, so as to have a third dimension study of cerebral angiograms. This has been done with injection of the internal carotid and vertebral arteries.

Both normal and pathologic cases were studied in this manner by using hypaque as the contrast medium.

Illustrations and descriptions of 12 cases studied are included in the article. The reproductions, although small in size, are in most of the cases excellent.

The authors state that the experience gained by addition of the submento-bregmatic view has encouraged them to utilize this method in future studies of intracranial tumors. The size and location of intracranial abnormalities provide the surgeon with additional information.—*Peter E. Russo, M.D.*

Neck and Chest

Murray, J. P. Neuromuscular and functional disorders of the pharynx. *J. Fac. Radiologists*, July, 1958, *9*, 135–141. (From: Sefton General and Alder Hey Children's Hospitals, Liverpool, England.)

Until recently, clinicians have seldom sought help from the radiologist in pharyngeal disorders, such as deglutition in myasthenia gravis, hysterical dysphagia, and pharyngo-esophageal incoordination, except to ask his aid in excluding a neoplasm or diverticulum. However, with the availability now of screen image intensification and cineroentgenography, more detailed information about pharyngeal function can be given. Fluoroscopy and roentgenography in the lateral projection usually provide adequate information, but examination in the anteroposterior projection is especially helpful in myasthenia gravis, and in the study of paralysis of specific muscle groups, as after poliomyelitis.

Myasthenia Gravis. Abnormalities of deglutition are seen in the *oral stage* with (a) hesitancy in initiating deglutition, (b) slow and progressively inefficient movements of the tongue, (c) inefficiency in forming a bolus on the back of the tongue, and consequently (d) measurable delay in clearing the mouth and oropharynx. In the *pharyngeal stage* of deglutition, abnormalities consist of (a) stasis in the pharynx and pooling in the pharyngeal recesses, (b) loss of tone and ballooning of pharynx on repeated swallowing, and inability to raise the larynx and so close off the upper pharynx, (c) eventual cessation of swallowing, and (d) nasal reflux. Disturbance of the oral stage tends to be associated with generalized myasthenia. In bulbar cases the pharyngeal dysfunction is the main cause of disability, and deglutition in such cases is very similar to that in progressive bulbar palsy in which there is persistent stasis in the pharynx. It is important to remember that pooling of contrast material in the valleculae may also occur in normal persons, especially in the older age groups. Cases of hysterical dysphagia may simulate the swallowing abnormality of myasthenia gravis, but these can usually be differentiated by the lack of response to prostigmin. A suitable dose of prostigmin as a diagnostic test is 1.5 mg. subcutaneously with 1/100 gr. atropine.

Hysterical Dysphagia. These cases fall into four groups: (1) Normal deglutition; (2) delay in initiating deglutition, with hesitant tremulous tongue movements; (3) inability to relax the upper constrictors to receive the bolus from the mouth, especially with dry solid foods; and (4) hypertonicity and prominence of the transverse part of the cricopharyngeus muscle.

Pharyngo-Esophageal Incoordination. The various sections of the gastrointestinal tract are separated from each other by sphincters, either anatomic or functional. The transverse belly of the cricopharyngeus apparently acts as the sphincter between the pharynx and esophagus. It relaxes to allow a bolus to pass down, also serving to prevent reflux of esophageal contents. The author refers to derangement of this function as *pharyngo-esophageal incoordination.* This is shown roentgenographically in incomplete relaxation of the cricopharyngeus as an indentation on the posterior aspect of the barium column as it proceeds through the postcricoid region. While the author believes that roentgenologic visualization of the cricopharyngeus muscle may occur normally, he states that demonstration of a prominent indentation by it should prompt a thorough investigation of the upper gastrointestinal or respiratory tract. In senile pharyngo-esophageal incoordination there is hypertrophy of the cricopharyngeus, associated with hypotonicity of the pharynx, pooling in the pharyngeal recesses, and often a spill over into the trachea. There is also a prominent anterior convexity of the lumen in the postcricoid region which may be mistaken for neoplasm. In patients at or after middle age the most difficult problem is to differentiate it from an early postcricoid neoplasm. In myasthenia gravis there is no hypertrophy or spasm of the cricopharyngeus and improvement after prostigmin is diagnostic. Incomplete relaxation of the cricogharyngeus is invariably present in recurrent laryngeal nerve palsy.—*Samuel G. Henderson, M.D.*

Madore, P., and Glay, A. Oesophagobronchial fistula complicating oesophageal diverticulum. *Canad. M. A. J.*, Oct 1., 1958, *79*, 564–566. (From: The Departments of Surgery and Radiology, St. Mary's Memorial Hospital, Montreal, Canada.)

Esophagobronchial fistula arising from neoplasm of bronchus or esophagus is a well-known entity. However, that produced from a traction diverticulum of the midportion of the esophagus, non-neoplastic type, is less frequent. One case is described in this article. The diagnosis was made with barium in the esophagus that outlined the fistulous tract to the bronchus. The patient's complaining symptom was massive hematemesis.—*F. B. Markunas, M.D.*

Watkins, David Hyder. Surgical complications in the thorax of staphylococcal pneumonitis. *A.M.A. Arch. Surg.*, Oct., 1958, 77, 508–537. (From: The Department of Surgery, University of Colorado and the Denver General Hospital, Denver, Colo.)

Staphylococcal pneumonia is far more common in infancy than in later childhood. Complication of empyema appears to occur inversely with age. This may be due to low concentrations of serum globulin in infancy. Frequently an organism from a purulent skin or subcutaneous lesion is responsible for the pulmonary infection. Pathologically, there is early necrosis of the alveolar walls with small abscess formation. These small abscesses tend to become confluent, and there may be rapid involvement of the pleural surfaces with ensuing emphysema. An abscess may also rupture into a bronchiole with the formation of a check-valve mechanism and a pneumatocele. This may be located subpleurally, and can rupture and cause a tension pneumothorax. Though the pneumatocele and emphysema are common complications, they are usually transient. Occasionally, though, a pneumatocele may require a period of six to nine months before it resolves.

The author gives 6 case histories in detail with accompanying roentgenograms. Surgical treatment with thoracotomy was necessary in several cases with rapid relief of the pleural complications. Other surgical procedures such as bronchoscopy and tracheostomy may also be required.

The author emphasizes that the primary therapy of staphylococcal pneumonitis is medical and that a wide variety of antibiotics may have to be used be-

cause of the increasing resistance of the organism. The complications which require surgical therapy are empyema, bronchopleural fistula, tension pneumothorax, fibrothorax, and occassionally abscess formation. With pleural complications, closed suction drainage is the treament of choice. Occasionally, decortication may be necessary for fibrothorax. —*Lawrence A. Davis, M.D.*

Pantone, Anton M. Radiographic chest examination of the tuberculous patient. *Radiology*, Sept., 1958, *71*, 336–339. (Address: 55 E. Washington St., Chicago 2, Ill.)

The author describes the method of roentgenographic chest examinations used in patients admitted to the Chicago State Tuberculosis Sanitarium. Routine posteroanterior and lateral films are obtained in all patients, utilizing the so-called high-kilovoltage technique: 125 kv. peak, with the ma.-sec. varying with the patient's size. These high-kilovoltage films exposed with the use of grid cassettes are thought to give a higher "yield" of lesions than conventional films using a lower kilovoltage range. Laminagraphy is employed in about 10 per cent of the patients with sections obtained in both anteroposterior and lateral views. This technique has been quite valuable in determining the presence or absence of disease in contiguous segments of the lobes such as in the superior segment of a lower lobe when the upper lobe is severely involved.

Bronchography, using dionosil oily, has been used in many patients to outline the bronchial tree. The rapid disappearance of the medium is a great advantage as the infiltrative lesions due to tuberculosis are not obscured and progress studies are not hindered. The method of this bilateral bronchography is described. Stereoscopic examinations of the chest are done but much less frequently than before as they seem to add little information, in the majority of cases, to that obtained by the routine high-kilovoltage posteroanterior and lateral roentgenograms. Apical-lordotic studies of the chest are performed very infrequently.—*Donald N. Dysart, M.D.*

Jacobs, Lewis G. Pulmonary torulosis. *Radiology*, Sept., 1958, *71*, 398–403. (Address: VA Hospital, Thirteenth and Harrison Sts., Oakland, Calif.)

A modified concept of torulosis of the lung is presented as a result of review of 10 cases. The solitary healed nodule was found to be the most common clinical form of pulmonary torulosis, occurring in 8 of the 10 cases. The pneumonia form of torulosis was present in 2 patients. All patients have remained well for periods of seven to forty-eight months following wedge resection or lobectomy.

The disease is usually a benign infection leading to the production of an inactive granuloma resembling a tuberculoma clinically, roentgenologically and pathologically. The significant finding in these granulomas is the organism itself, although it generally is impossible to obtain cultures from the "healed" lesions. Severe acute forms of the disease, with high mortality rates appear to be less common. Generalized torulosis, *Torula* meningitis, pneumonic or granulomatous torulosis of lung or combination of these lesions are serious forms of the disease which generally do not respond to any form of treatment. Spontaneous remissions may occur, but even in such cases the mortality rate approximates 60 per cent.—*Arno W. Sommer, M.D.*

Melamed, Myron. Roentgen evaluation of the chest after thoracic surgery. *Radiology*, Sept., 1958, *71*, 348–349. (Address: 55 E. Washington St., Chicago 2, Ill.)

Because of the increasing number of surgical procedures involving the thorax, the interpretation of the postoperative chest roentgenogram has become more complex. To aid in the evaluation of such roentgenograms, the author offers a classification which outlines the postoperative changes which may appear in the various structures of the thorax. These changes include those in the soft tissues, ribs, diaphragm, mediastinum, pleural cavity and lung parenchyma. The possible complications are also classified. This classification was based on a study of 300 selected surgical procedures involving the thorax performed principally for pulmonary tuberculosis. The procedures used were: segmental resection, lobectomy, pneumonectomy and various space-diminishing procedures.—*Z. Petrany, M. D.*

Oderr, Charles P., Pizzolato, Philip, and Ziskind, Joseph. Emphysema studied by microradiology. *Radiology*, Aug., 1958, *71*, 236–245. (Address: C. P. Oderr, 1601 Perdido St., New Orleans 12, La.)

The authors present a significant contribution to the study of emphysema with a microradiologic evaluation of the lungs of 65 nearly consecutive autopsies on male veterans. Lungs which can be removed intact are severed at the bronchus near the carina and perfused with air at normal inflation pressure (usually 15 to 20 cm. of water) for several days until thoroughly dry. The lung is then roentgenographed as a whole after which it is cut into slices of varying thickness. These slices are then studied with beryllium-window-tube roentgen-ray machines with kilovoltages from 3 to 15. The technical factors are presented in tabular form. After study of the roentgenograms, blocks of tissue are removed for microscopic analysis.

Examination of the normal lungs involved in this study substantiates previous observations that the vascular patterns of the central or medullary portions are distinctly different from the peripheral or cortical portions of the lung. In the central zone the

arterial and venous trunks are widely separated, while in the peripheral zone the arterioles and venules closely approximate one another. It is suggested that this difference in zonal anatomy may have physiologic and pathologic significance.

The cases of emphysema were found to be of three general types: those with principal involvement of the central zone; those with principal involvement of the peripheral zone; those in which zonal localization does not exist and there is marked associated interstitial fibrosis. Microradiographs of each type are presented. The central zone type of emphysema is encountered most frequently and, unless the disease is far-advanced, routine roentgenographic and fluoroscopic studies do not show signs of emphysema. The peripheral type of emphysema is second in frequency and seems to develop from the immediate subpleural area. The non-zonal type of emphysema is least frequently encountered and seems to be of different character since it is associated with interstitial fibrosis which frequently is demonstrated on the clinical roentgenogram. Most of the cases studied showed minute granuloma-like pulmonary lesions, the role of which is uncertain at present.

The authors conclude that the major factor in the production of emphysema is expiratory bronchiolar collapse due to an unfavorable shift of the air pressure on the inside and outside of the bronchiolar wall during expiration. This may be aided by mucus cohesion, spastic narrowing, or diminished elastic recoil due to fibrosis.—*Edward B. Best, M.D.*

Lübker, Heinz. Zur Technik der Schichtaufnahmen des Bronchialbaumes. (A contribution to the laminagraphic technique of the bronchial tree.) *Röntgen Blätter*, Dec., 1958, *11*, 281–284. (Address: Oberfeldwebel an der Röntgenabteilung des Bundeswehrlazarettes, Giessen, Germany.)

Because of the relatively high radiopacity of the osseous and mediastinal structures, a hard exposure is necessary in order to obtain good definition of the trachea and main stem bronchi. This in turn causes excessive film density in the region of the smaller bronchi situated in the lung parenchyma.

Trying to artificially simulate the situation as present in pulmonary atelectasis when roentgenograms of uniform density are commonly obtained, filtration of the section of the beam traversing the "radiolucent" lung parenchyma was attempted and was found to give very good results.

The author utilizes copper and aluminum sheets of 14×14 inches which are placed on a small four-legged table-like wooden structure between the patient and the tube. Proper localization of the filter in relation to the lung field may be accomplished fluoroscopically.

After having determined the desired depth, preliminary exposures, 1 without filter and 3 with changing filtration of 2 mm. Al, 3 mm. Al, and 0.3 mm. Cu are taken and the desired film density then selected. With some experience these test exposures will prove superfluous and the correct filter will be chosen after inspection of the conventional chest roentgenogram alone.—*Walter G. Heiman, M.D.*

Bonte, Frederick J., and Reynolds, Jack. Hydrocarbon pneumonitis. *Radiology*, Sept., 1958, *71*, 391–397. (Address: F. J. Bonte, 5201 Harry Hines Blvd., Dallas 19, Texas.)

One of the leading causes of accidental death in the very young is poisoning. Bain has stated that 25 per cent of all poisonings in children under the age of five are due to the consumption of petroleum products, especially kerosene. Others include furniture polish, lighter and cleaning fluid, insecticides and similar materials containing hydrocarbons. Clinically, the outcome is almost always favorable. The finding of chief interest to the radiologist is the rapid development of a pneumonitis of rather typical appearance which may be appreciable on roentgenograms made as early as twenty minutes after ingestion and may persist for weeks after symptoms have subsided. Of the various mechanisms of production of this infiltrate, aspiration of the toxic agent is considered to be the most likely one.

The authors report a study of 65 such patients admitted to the emergency room of the Parkland Memorial Hospital in Dallas, Texas in the four year period between 1952 and 1956. Of these, 60 had at least one chest roentgenogram, and only these are considered in the present report. The findings on the initial posteroanterior chest roentgenograms of these patients were evaluated according to Foley's classification which is as follows: Group I, infiltrate involving 10 per cent of lung area; Group II, 10–30 per cent of lung area involved; Group III, more than 30 per cent of lung area involved. The results are tabulated and it was found that the majority of cases fall into Groups I and II. Only 3 cases fell into Group III; 4 cases showed questionable involvement and 4 cases showed no evidence of an infiltrate on one or more chest examinations. The infiltrates ranged in character from softly flocculent to homogeneous. Three typical distributions of the infiltrate could be seen. The most common was bilateral basal. The least common was in the right base only. The third manifestation was perihilar infiltrate and clear bases, suggesting an interstitial process. This latter is referred to as the "edema" type. Typical roentgenograms are presented to show these variations.

There is a direct relationship between the amount of ingested material and the amount of infiltrate seen on roentgenograms. There is no correlation between a history of vomiting and the extent of the lung involvement. Characteristically, resolution of the

roentgenographic findings lags well behind clinical improvement. Development of pleural effusion is uncommon and has been attributed to secondary infection. It is impossible to distinguish between the various hydrocarbon poisons on the basis of the appearance of the pulmonic infiltrates they produce.

The authors also report on experimental studies of hydrocarbon pneumonitis produced in dogs by instillations of kerosene through a catheter into the lung, in order to determine the minimum interval at which an infiltrate could be detected and its rate of development following the instillation.—*Z. Petrany, M.D.*

NELSON, SIDNEY W., and CHRISTOFORIDIS, ANTHIMOS. Reversible bronchiectasis. *Radiology*, Sept., 1958, *71*, 375–382. (Address: S. W. Nelson, Department of Radiology, Ohio State University, Columbus 10, Ohio.)

Bronchiectasis in its early stages is frequently a reversible process, particularly in children and young adults following acute pneumonitis and atelectasis. If intensive medical therapy is successful in alleviating the infection before the occurrence of destructive changes in the musculo-elastic tissues of the bronchial wall, the involved bronchi will return to normal.

Careful and thorough bronchography is the most reliable and practical diagnostic method for detecting bronchiectasis and repeated bronchograms should be done in order to follow the course of the disease. There are a number of signs which indicate reversibility. When the mucous membrane and the musculoelastic elements are intact, during the act of cough a swift peristaltic wave runs from the small bronchi toward the trachea. If these structures are destroyed, the peristaltic wave cannot originate in the bronchi and the cough is ineffective. This sign, although helpful, is difficult to study and is not absolute. Another sign which is usually seen in the reversible cases of bronchiectasis is a varicose and fusiform appearance of the dilated bronchi. In these types of the disease, narrowing at the origin of the segmental bronchi or of the third or fourth bronchi indicates that the circular and muscular elements are still present. The most reliable evaluation of reversibility is made by serial bronchography with the presently available quickly eliminated contrast media.

The authors report 4 cases of bronchiectasis which demonstrate complete disappearance of bronchiectasis which had been clearly evident on the previous examination.—*Arno W. Sommer, M.D.*

PFUETZE, KARL H., and PYLE, MARJORIE M. Medical management of pulmonary tuberculosis. *Radiology*, Sept., 1958, *71*, 340–344. (Address: K. H. Pfuetze, 1919 West Taylor St., Chicago 12, Ill.)

The authors discuss the presently accepted general principles involved in the treatment of pulmonary tuberculosis, the regimens most commonly used, and the results.

The three major antimicrobial agents employed today in the treatment of tuberculosis are isoniazid, streptomycin (and its hydrogenated derivative, dihydrostreptomycin) and para-aminosalicylic acid (PAS). Though there is no generally accepted "optimum" chemotherapy regimen, the following are the most commonly used: (a) isoniazid, 100 mg. t.i.d. plus streptomycin, 1.0 gm. twice weekly; (b) isoniazid 100 mg. t.i.d. plus PAS, 12 gm. daily; (c) isoniazid, 100 gm. t.i.d. plus streptomycin, 1.0 gm. twice weekly, plus PAS, 12 gm. daily in 3 or 4 doses. Chemotherapy should be uninterrupted and prolonged—for at least one year and often for two or more years. In some cases chemotherapy should probably be given indefinitely. The most satisfactory results are obtained in patients with lesions of recent origin including those with cavitation. The older the process and the greater the component of caseation and cavity formation the less is the likelihood that chemotherapy alone will result in satisfactory healing. The more frequently chemotherapy has been interrupted in the past, the poorer the prognosis and the greater the chance that drug-resistant organisms will emerge. In retreatment cases the sensitivity of the organisms must be determined.

Rest is still important in the treatment of pulmonary tuberculosis, particularly during the early active stage of the disease. A few carefully selected patients may be treated in the home but in the majority of the cases the facilities and trained personnel of the tuberculosis hospital provide the best means for a rapid and permanent recovery.—*W. M. McBride, M.D.*

LANGSTON, HIRAM T. The surgery of pulmonary tuberculosis. *Radiology*, Sept., 1958, *71*, 345–347. (Address: 1919 West Taylor St., Chicago 12, Ill.)

Surgical intervention is indicated in pulmonary tuberculosis when demonstrable residuals of lung destruction appear that cannot or do not respond to antibiotics and chemotherapeutic agents. These residuals include open cavities, conglomerate and large focal areas of necrosis (1.0 to 1.5 cm.), bronchiectasis, and carnification due to organization of pneumonic exudates. Prolonged medical observation of the above conditions is not advised in view of the fact that the commonly used drugs provoke the resistance of the tubercle bacillus, and that the results of resective surgery are poor except when carried out under the protection of chemotherapy. Therefore, the ideal time for surgical intervention is that moment when this form of treatment is deemed necessary and when chemotherapeutic control is achieved, a state evidenced by negative sputum examinations and roentgenologic stability of the lesion.

Current surgical philosophy favors resection over collapse procedures which, in the present era, means

thoracoplasty. Even segmental resections are safe and satisfactory but, in the presence of meager or poor chemotherapeutic control, wider resections to the extent of lobectomy or pneumonectomy are less prone to develop tuberculous complications. Collapse procedures, however, still are indicated in those instances when the amount of lung tissue requiring removal will compromise the cardiorespiratory reserve, and when the safety of the procedure is doubtful due to inadequate chemotherapeutic control. A combination of resection and collapse is used when adequate resection would leave a lung residual too small to fill the hemithorax. Both procedures may be done at the same time but the author favors a two-stage procedure with resection preceded by thoracoplasty.

The author reports a series of 423 operations for pulmonary tuberculosis with a mortality rate of 3.7 per cent in pneumonectomies and 1.7 per cent in lobectomies. Relapses occurred in approximately 3 per cent of the surgical patients treated.—*Edward B. Best, M.D.*

Roth, M. Zur Frage der Flüssigkeitsresorption aus der Lunge; vom Gesichtspunkt der bronchographischen Kontrastmittel. (Fluid absorption from the lung, as regards bronchographic contrast substances.) *Fortschr. a. d. Geb. d. Röntgenstrahlen und d. Nuklearmedizin*, Nov., 1958, *89*, 517–527. (Address: Centr. rtg. FN, Pekařská 53, Brno, Czechoslovakia.)

In spite of progress made during the last years, the problem of finding a biologically adequate and harmless bronchographic contrast medium remains unsolved. The water soluble substances, although having greater advantages, leave behind in the alveoli a not absorbable cellulose derivative used as a viscous carrier which does as great damage as the iodized oil.

The author reported in 1956 of his experimental use of dried blood plasma as a viscous carrier for bronchographic contrast media. The rationale is that blood plasma is the body's own antigen free, fermentatively attackable and partly absorbable substance which, even when it remains for a long period in the pulmonary parenchyma, produces no changes. However, the disadvantage inherent to the instillation of a hypertonic solution in the pulmonary parenchyma remains equal to that of other water soluble contrast media.

The author demonstrates roentgenographically the result of such instillations. One to two minutes after the injection of 2.5 ml. of a hypertonic contrast medium (ultraren-Na or diodon 50 per cent) through a rubber catheter in the periphery of the lower lobe of a rabbit, the metallic density starts to diminish and in five minutes disperses into a not quite homogeneous area of increased tissue density in which the metallic component can no longer be identified. This area grows in size and density and reaches its high point after approximately two hours. It starts receding at the sixth hour and the lung fields return to their normal appearance after sixteen to twenty hours. These changes are explained on the ground of osmotic drawing of fluid from the ample surrounding vascular bed by the hypertonic contrast solution. The metallic density is thinned due to absorption. The increased tissue density is the result of pulmonary edema. The same area of increased tissue density in two hours and its disappearance in sixteen hours is produced after the instillation of 2.5 ml. of 5 per cent salt solution without contrast substance, indicating that it represents lung edema. No similar areas of increased tissue density occur after the instillation of distilled water or physiologic salt solution in the lung parenchyma. Subcutaneous and intramuscular injections of contrast substance were made and it was found that their absorption is much slower than in the lungs due to the greater vascularity in the lung parenchyma and, therefore, the more rapid osmotic equalization.

The author compares the absorption of the diluted plasma contrast substance in the lung periphery to the lysis of a pneumonic exudate.—*Salomon Silvera, M.D.*

Rowe, Caroline W. Increasing scope of bronchography with dionosil. *Radiology*, Sept., 1958, *71*, 383–390. (Address: Medical Branch, University of Texas, Galveston, Texas.)

An analysis of 400 bronchographic examinations using dionosil at the John Sealy Hospital is presented. This rapidly absorbable medium has markedly increased the usefulness of bronchography in the diagnosis of lung pathology. A satisfactory bronchogram (complete visualization of the peripheral bronchioles as well as of the larger bronchi) was obtained in 75 per cent of the cases. In one-fourth the filling was incomplete and re-examination was required. Among the reasons for incomplete visualization were failure to use sufficient dionosil, improper preparation of the dionosil, bronchospasm, obstruction from secretions or tumor, and unsatisfactory anesthesia. In order to avoid the above, the medium should be thoroughly shaken prior to its use; bronchodilators may be administered where bronchospasm has occurred; and postural drainage prior to re-examination may help to remove obstructions due to secretions.

Reactions to the oily dionosil were noted to be few although the use of aqueous dionosil was found to produce a fairly high incidence of a "flu-like" syndrome in the twenty-four hour period following examination. This is believed due to the emphasis on complete peripheral bronchiolar filling. The author now prefers the oily suspension as reactions of this nature are reduced with this medium.

The increasing value of bronchography and its many indications as well as the additional informa-

tion obtainable when compared with conventional chest roentgenography are stressed. A comparison of diagnoses as determined by chest roentgenograms and the additional information gained by bronchography is made with the final diagnoses in 200 abnormal bronchograms. This is arranged in table form. Accompanying illustrations demonstrate the efficacy of repeating examinations when complete peripheral filling is not obtained.—*John S. Alexander, M.D.*

Lodwick, Gwilym S., Keats, Theodore E., and Dorst, John P. An evaluation of the significance of transverse hilar measurements in the diagnosis of primary lung cancer. *Radiology*, Sept., 1958, *71*, 370–374. (Address: G. S. Lodwick, School of Medicine, University of Missouri, Columbia, Mo.)

In a study of the roentgen characteristics of 541 cases of clinically or pathologically diagnosed primary lung cancer, complete hilar measurements were obtained in 370 cases in an attempt to evaluate the usefulness of previously established criteria of hilar measurement in the detection of lung cancer. Two separate hilar measurements were made in each case: one from the point of bifurcation of the pulmonary artery on either side, representing the least hilar measurement; and the other from the point farthest out on the hilus on either side, representing the greatest hilar measurement. In the final analysis the greatest hilar measurement was used because it probably closely approximated hilar measurements previously reported by other authors. The cases were also analyzed according to the location of the primary lung cancer in order to evaluate the influence of location upon diagnosis of the lesion or survival of the patient.

In considering all cases of carcinoma of the lung, regardless of location, it was found that the total transverse hilar measurement was greater than normal in 31.9 per cent of the cases, and that the unilateral hilar measurement was greater than normal in 36.8 per cent. Conversely, in from 63 to 68 per cent of all cases of known primary lung cancer, the most generous hilar measurements are within the range of normal. In addition, 13.5 per cent of the known cases of lung cancer had total transverse hilar measurements of less that 11.0 cm., a range said to exclude the presence of cancer of the hilus. In the group of very early lung cancers, where the tumor is not readily apparent in the roentgenogram, and where hilar measurements could be of greatest help in making a diagnosis, these measurements were abnormal in only 19.4 per cent.

The importance of unilateral hilar enlargement as an early sign of lung cancer is not to be minimized. Normal hilar measurements by no means exclude a clinically detectable neoplasm, or even an enlarged hilus.—*Walter H. Jarvis, Jr., M.D.*

Punchetti, V., Ionescu, L., and Cubillos, L. Gastroenterogene Mediastinalcysten. (Gastroenterogenic mediastinal cysts.) *Thoraxchirurgie*, Nov., 1958, *6*, 251–261. (From: Chirurgische Klinik der Medizinischen Akademie, Düsseldorf, Germany.)

Among 64 patients, who were operated on for mediastinal cysts at the surgical department of the Medical Academy in Düsseldorf between 1947 and 1957, cysts of gastroenterogenic origin were found in 3 cases. The diagnosis was proved by histologic examination.

The case reports of the 3 patients are given and the etiology, symptomatology and complications are discussed. The authors include an excellent review of the world literature on the gastroenterogenic cysts.—*Eckart Schackow, M.D.*

Steinberg, Israel, and Sammons, B. P. Aneurysmal dilatation of the aortic sinuses in coarctation of the aorta: Report of two new cases and review of the literature. *Ann. Int. Med.*, Oct., 1958, *49*, 922–934. (From: The Departments of Radiology, The New York Hospital-Cornell Medical Center, and the U. S. Naval Hospital, St. Albans, N. Y.)

The authors present 2 cases of aneurysm of the aortic sinuses associated with coarctation of the aorta to be added to the 5 cases previously reported in the literature. Such aneurysms are apparently more common than previously suspected. Five cases associated with arachnodactyly have also been reported.

The aortic sinuses are three small dilatations in the wall of the aorta immediately above the valves. Each one lies just above a valve cusp. The right and left coronary arteries usually arise from the right and left aortic sinuses. The posterior sinus is not associated with a coronary artery. The aortic sinuses are intracardiac and cannot be identified on conventional roentgenologic studies.

Edwards and Burchell believe that in aortic sinus aneurysms the wall is made up of the atrial wall instead of the aortic media. The pressure in the aorta causes bulging in the weak atrial wall. In some cases of Marfan's disease medionecrosis of the aortic wall occurs just above the heart to allow the dilatation.

The ages of the patients with aortic sinus aneurysm associated with coarctation of the aorta varied from thirteen years to twenty-seven years with an average age of twenty-one years. Some of the cases were asymptomatic. Systolic murmurs transmitted to the axilla and back were common. The signs and symptoms of coarctation of the aorta were present in practically all cases.

All the sinuses are usually involved with rarely only one being involved. Angiocardiography is the only accurate diagnostic procedure. When a sinus

ruptures there is a sudden, severe dyspnea, chest pain, right heart overloading and a machinery type murmur.

The prognosis is not necessarily bad particularly when the associated coarctation is corrected. The reduction in the blood pressure certainly postpones rupture and may prevent it. These patients may return to normal occupation and practically normal athletic activities.—*Frank M. Windrow, M.D.*

Holman, Cranston W., and Steinberg, Israel. The role of angiocardiography in the surgical treatment of massive pericardial effusions. *Surg., Gynec. & Obst.*, Nov., 1958, *107*, 639–647. (From: The Departments of Surgery and Radiology, The New York Hospital-Cornell Medical Center, New York, N. Y.)

The diagnosis of pericardial effusion is frequently difficult even with all the conventional tests. Angiocardiography is an accurate diagnostic method for pericardial effusion as it outlines the cardiac chambers and clearly permits recognition of the surrounding fluid. Because conservative therapy including paracentesis is frequently prolonged and sometimes ineffective, surgical therapy is becoming more common. It usually leads to a prompt, permanent cure and sometimes establishes a difficult diagnosis such as tuberculous pericarditis, pericardial cyst or tumor. The procedure of choice is surgical drainage and establishment of a pericardial pleural window for drainage.

Angiocardiography has shown that pericardial effusions usually begin in the diaphragmatic portion of the pericardium and later extend into the retrosternal portion. They displace the heart backward but do not invade the posterior pericardium.—*Frank M. Windrow, M.D.*

Lull, George F., Jr. Pericardial celomic cyst; a re-evaluation. *Radiology*, Oct., 1958, *71*, 534–541. (Address: Fitzsimons Army Hospital, Denver 8, Colo.)

The author presents a review of 15 cases of pathologically proved pericardial celomic cysts and enumerates the other mass lesions occurring in the anterior inferior mediastinum that have to be considered in the differential diagnosis. Roentgenographically all the pericardial celomic cysts have the appearance of cystic lesions. All showed evidence of demarcation of the lesion from the cardiac shadow. A fairly sharp angle at the border of the area of density where it merges with the heart suggests that the mass is probably separate from the heart even though it lies immediately adjacent to it.

An alteration in size and shape of the lesion on inspiration and expiration is thought to indicate nothing more than a large cyst which could be of any etiology. Variation in contour is governed by the size of the cyst, the tension of the fluid contents, and the relationship to surrounding structures. Loculation of a cyst does not appear to aid in differentiating between pericardial celomic cysts and lymphangiomatous cysts as both may be multilocular or unilocular.

The etiology of these pericardial celomic cysts is theorized as being two-fold. First, the potential for the development of a cyst occurs during embryologic growth where there is a persistence, as a separate cavity, of one of the mesenchymal lacunae which fuse to form the pericardial celom. Second, a trigger mechanism which accounts for the time of appearance of these lesions and for their roentgenographic appearance is necessary. This mechanism must be some form of injury, either an inflammatory disease or actual trauma. This theory probably explains why 12 of 15 cases in this series had had prior negative chest roentgenograms or negative reports. In most cases symptoms do not seem to develop until the cyst becomes large enough to produce effects by pressure or because of the location.—*Donald N. Dysart, M.D.*

Ciarpaglini, L., and Iannaccone, G. Dynamische Veränderungen des varikösen Ösophagus; Röntgenkinematographische Untersuchung mit dem Bildverstärker. (Dynamic changes of the varicose esophagus; cineroentgenographic examination with the image amplifier.) *Fortschr. a. d. Geb. d. Röntgenstrahlen und d. Nuklearmedizin*, Nov., 1958, *89*, 551–557. (Address: G. Iannaccone, Istituto di Radiologia dell'Università-Policlinico, Roma, Italy.)

In addition to the conventional roentgenographic examination of the esophagus, the authors used cineroentgenography to study the various phases of dynamics during the passage of the contrast medium in the presence of varices. Eleven cases of esophageal varices secondary to intra or extrahepatic block were examined.

A Phillips image amplifier, 12 cm. in diameter, and a 35 mm. camera were used. The technique employed was 86–94 kv., 12 ma., and a speed of 22 frames per second. The contrast medium administered was a rather thick barium mixture. Neither the Valsalva and Müller maneuvers, nor the forceful inspiration or expiration, were of any value to more adequately visualize the condition. The varices were best seen when the esophagus was partially filled and in the rest phase. The appearance of the involved segment changed completely from one moment to another in the same individual. Therefore, a classification into various stages (Robert and Hoffman) or into different types (Papillon *et al.*) based on the roentgen appearance is not justifiable.

In general, the dynamic changes in a varicose esophagus are the result of the different aspects of dyskinesia when the latter is understood to mean any disturbance of the tone and motility. Decrease of the esophageal tone down to complete atonia in the varicose segment and sometimes in the entire esophagus has been noted which at times can assume the dimensions of a true megaesophagus. In addition there is a characteristic delay in the so-called "cleansing" of the esophagus due to retardation of the contrast medium in the crypts between the varicose knots. A third observation in some patients was the presence of anomalous contraction of the esophageal walls in the form of fine wave movements which do not propel the esophageal contents and which were named "tertiary peristalsis" or esophageal "rippling."—*Salomon Silvera, M.D.*

BALOW, R. M., and WIETERSEN, F. K. Postemetic rupture of the esophagus; a report of three cases. *Radiology*, Sept., 1958, *71*, 420–424. (Address: R. M. Balow, Grace Hospital, 18700 Meyers, Detroit 26, Mich.)

Post-emetic rupture of the esophagus, or spontaneous rupture of the esophagus, is a rather uncommon accident, and although the history, clinical, and roentgen findings are characteristic, the diagnosis is frequently missed. The condition usually terminates fatally without prompt surgical treatment. The rupture is generally considered to be the result of increased intraluminal pressure, due to spasm of the pyloric and cricopharyngeus muscles with associated vomiting and retching. The tear involves all layers—the outer layers first and the mucosa last. It always develops in a vertical direction and may be a few millimeters to 8 cm. in length. It is usually in the distal portion on the left posterolateral aspect just above the diaphragm because of inherent weaknesses at this level. The closer relationship of the left pleura to the mediastinal portion of the tear explains the high frequency of left-sided hydropneumothorax. The reported cases, predominantly males (5 to 1), with an average age of forty-five to fifty years, often give a history of overindulgence in eating and/or drinking followed by vomiting and the sudden onset of severe pain in the epigastric or substernal area. The patient becomes critically ill and the course is rapidly downhill. Physical findings will vary depending on the time elapsed following the rupture, and may include restlessness, dyspnea, cyanosis, a nasal twang to the voice, subcutaneous emphysema about the neck and upper chest, evidence of unilateral or bilateral hydropneumothorax, and upper abdominal rigidity. Sooner or later the patient goes into shock due to pain, contamination, and anoxia.

The usual roentgenographic findings are: mediastinal emphysema; subcutaneous emphysema; and pneumohydrothorax, usually on the left (7 per cent on the right; 28 per cent bilateral). A lipiodol swallow will as a rule demonstrate the fistulous tract. No case has been reported with free air below the diaphragm. Among the conditions to be differentiated are perforated peptic ulcer, acute pancreatitis, empyema of the gallbladder, acute coronary occlusion, pulmonary embolism, and spontaneous pneumothorax. The possibility of esophageal rupture must be kept in mind, and by correlation of the roentgen observations with other findings, even in the atypical case the diagnosis can be made. Once the diagnosis has been confirmed, rapid preparation for surgery is necessary. The critical period appears to be about twelve to fifteen hours, after which the mortality rises markedly. If at all possible, surgery offers the best hope of survival, and if treatment is undertaken early, the response has often been dramatic, with marked improvement of the general condition and eventual recovery. Three cases are presented, all of which ended fatally.—*Walter H. Jarvis, Jr., M.D.*

ABDOMEN

GLENDINNING, A. C. Barium meal and follow-through. *Lancet*, Sept. 27, 1958, *2*, 664–667. (From: Bromely Group of Hospitals, Kent, England.)

The author reports on 1,000 consecutive barium meal and follow-through examinations. The follow-through examinations were made at one hour after ingestion and again at six hours.

The barium meal examination was of great value. Abnormalities were found in the upper gastrointestinal tract and related structures in 55.6 per cent of the patients. Slow emptying of the stomach was usually due to duodenal abnormalities.

The barium follow-through examination was often time-consuming and of no value. It is not a substitute for a proper small bowel examination or a barium enema study.—*Frank J. Rigos, M.D.*

ELLIS, KENT. Gastrojejunal ulcer. *Radiology*, Aug., 1958, *71*, 187–196. (Address: 622 W. 168th St., New York 32, N. Y.)

Of 238 patients with a clinical diagnosis of gastrojejunal ulcer there were 134 cases considered to be proved gastrojejunal ulcers; 56 of these followed partial gastrectomy, and 78 occurred after simple gastrojejunostomy. In 120 of the 134 patients the original diagnosis was duodenal ulcer; in 2 patients both gastric and duodenal ulcers were present, and in 12 patients the site of the original peptic ulcer was unknown. Of the 134 patients 115, or 86 per per cent, were men. Gastrointestinal bleeding occurred in 69 per cent. Although most patients had moderate to severe abdominal pain, none was noted in 11 per cent. The incidence of bleeding prior to original surgery and the incidence of more than one previous operation for peptic ulcer was approximately twice as great in the post-partial gastrectomy group as in the post-gastroenterostomy group.

There appeared to be a longer average period of observation following a simple gastroenterostomy.

In this study, 45 of 56 (80 per cent) of the proved gastrojejunal ulcers occurring in the post-partial gastrectomy group of patients were shown on the films; in the post-gastroenterostomy group 44 of 78 (56 per cent) of the ulcers were demonstrated. The majority of the ulcers recognized were 5 mm. or more in diameter. In 3 cases huge ulcer craters were well demonstrated but not recognized as ulcers by the initial examiner, presumably because of their unexpected size. Multiple gastrojejunal ulcers were present in 6 cases. The chance for positive roentgenologic diagnosis of gastrojejunal ulcer after simple gastroenterostomy is distinctly less than after partial gastrectomy. As in the diagnosis of all peptic ulcers, the most important roentgen finding is the niche representing the actual ulcer crater. Roentgenologically the ulcers appeared to be either at the stoma or more often in the efferent jejunum opposite the stoma. Further analysis of the results of this study revealed that in those patients who had a reasonably adequate upper gastrointestinal series within two weeks prior to gross pathologic demonstration of a gastrojejunal ulcer 5 mm. or more in diameter, the ulcer could be demonstrated on the films in 24 of 26 cases following partial gastrectomy, and in 37 of 54 cases following simple gastrojejunostomy. In those patients who had both upper gastrointestinal tract hemorrhage and moderate to severe abdominal pain the ulcer could be demonstrated in 22 of 24 post-partial gastrectomy patients and in 17 of 22 post-gastroenterostomy patients. The most important ancillary signs of gastrojejunal ulcer are as follows: obstruction of a previously patent anastomosis or jejunal loop; spasm or edema with narrowing of the jejunum, associated with effacement of the jejunal mucosal folds; radiation of the mucosal folds from the region of the small ulcer crater, and tenderness over the stoma. The technique of the examination is described.

Although chronic subacute or acute perforation is characteristic of the majority of these ulcers for which operation is performed, free intraperitoneal perforation and perforation into the colon are of special importance. Gastrojejunocolic fistula cannot be excluded until after a barium enema examination is performed. Obstruction of the stoma or the jejunum may occur secondary to either acute or chronic inflammatory changes associated with gastrojejunal ulcer. Active duodenal ulcer is extremely rare in the presence of a well functioning stoma. The precise importance and incidence of superficial gastric or jejunal ulceration and gastrojejunitis are indeterminate, but must be considered in the differential diagnosis. False positive identification of craters is a serious problem. Pouch-like deformities on the gastric side of the anastomosis are common. Pseudodiverticula may persist in the jejunal loop after gastrojejunal ulcer healing or may be the consequence of previous surgery. While obstructive symptoms may be caused by gastrojejunal ulcer, high intestinal obstruction may occur in the postoperative duodenal ulcer patient secondary to other conditions. Carcinoma of the stomach may occur, especially in the post-simple gastrojejunostomy patient, but rarely develops at the stoma. The possibility that more remote organs or systems are producing the patient's symptoms must always be considered, especially when no gastrojejunal ulcer crater is visible roentgenologically.—*Walter H. Jarvis, Jr., M.D.*

Stein, Joseph, Perlman, Bernard B., and Povalski, Alexander. Gastroduodenal intussusception. *Radiology*, Sept., 1958, *71*, 412–414. (Address: J. Stein, VA Hospital, 130 W. Kingsbridge Road, Bronx 68, N. Y.)

A case of gastroduodenal intussusception in a sixty year old white male is reported because of its relative rarity. At surgery a large sessile polyp, measuring 4 to 5 cm. in diameter, was found prolapsed into the second and third portion of the duodenum with the pars media of the stomach intussuscepted into the pylorus and duodenum. The case had been diagnosed preoperatively as a tumor, but intussusception was not recognized.

The criteria for the diagnosis of gastroduodenal intussusception are: (a) a space-occupying mass which changes its anatomic position in relation to the stomach and duodenum, thus establishing prolapse; (b) a concentric appearance of the intussuscipiens containing the intussusceptum.—*Arno W. Sommer, M.D.*

Soteropoulos, Constantine, and Gilmore, John H. Roentgen diagnosis of acute appendicitis. *Radiology*, Aug., 1958, *71*, 246–256. (Address: C. Soteropoulos, Illinois Masonic Hospital, 834 Wellington Ave., Chicago 14, Ill.)

The authors report the results of a three year study undertaken to evaluate the possible usefullness of the plain film abdominal survey in the diagnosis of acute appendicitis. Twenty-five cases of acute appendicitis were studied roentgenographically with detailed analysis of the surgical, pathologic, and roentgenologic findings.

The most important single sign was a fluid level in the cecum, which is best seen in the left decubitus position, and which is occasionally associated with small fluid levels in the terminal ileum. Other roentgen findings listed in the minimum survey study which includes a supine, upright, and right or left decubitus views were: (1) coarsening and edema of the mucosa of the cecum; (2) localized ileus of the terminal ileum; (3) fecaliths of the appendix in the presence of suggestive clinical findings; (4) localized ileus of the loops of the terminal ileum; (5) widening and shortening of the extraperitoneal fat line;

(6) spasm and blurring of the distal portion of the right psoas muscle with secondary scoliosis.

It is concluded that the plain film survey of the abdomen has definite value in the diagnosis of acute appendicitis, especially in those cases with atypical clinical features.—*W. M. McBride, M.D.*

DUBRA, CARLOS CÉSAR. Coleperitoneo enquistado. (Encysted choleperitoneum.) *Prensa méd. argent.*, March 28, 1958, *45*, 1361–1366. (Address: French 2331, Buenos Aires, Argentina.)

Biliary peritonitis, choleperitoneum and biliary ascites represent definite clinical entities. Biliary peritonitis is an acute peritonitis caused by bile in the peritoneal cavity. Choleperitoneum may be acute or chronic, of which the latter is localized or cystic. Biliary ascites is a somewhat equivocal term. In this condition, ascites appears because of leakage of bile into the peritoneum or a preexisting ascites becomes tinted by bile.

The presence of bile in the peritoneum can be attributed to 3 basic causes: (1) pathologic conditions of the biliary tract such as calculi, neoplasm and biliary pancreatitis; (2) trauma of the liver; and (3) miscellaneous surgical causes. Shock is a prominent symptom in internal hemorrhage associated with rupture of the liver and requires immediate surgical intervention. The presence of choleperitoneum becomes evident later.

There is no satisfactory explanation for the mechanism of shock in choleperitoneum. Four factors (toxic, infectious, circulatory and mechanical) each may play a part.

The diagnosis of choleperitoneum is not easy. In the initial period, the symptoms of choleperitoneum are difficult to distinguish from the disease which induced it. The presence of a large epigastric tumor which produces little pain, with or without jaundice in a patient who has suffered from an acute injury; surgical intervention on the biliary tract; or an acute abdominal condition will require consideration of hematoma of the omentum, subphrenic abscess, pseudocyst of the pancreas and hydatid disease. Choleperitoneum should also be included in the differential diagnosis.

Laboratory data may be helpful. Anemia, elevated sedimentation rate, hyperbilirubinemia, or bile in the urine are usually present. Roentgenologic examination will establish the position of the tumor.

Treatment consists primarily of surgical drainage of the cavity above the location of the tumor.—*Murray M. Friedman, M.D.*

GRAY, E. DUFF. (Manchester, England.) Some radiological aspects of biliary tract disease. President's address. *Proc. Roy. Soc. Med.*, Oct., 1958, *51*, 793–796.

The author reviews the subject of cholecystography and some of the anatomic and physiologic aspects of the biliary tract and related organs. Three current contrast media are discussed (pheniodol, telepaque and biligrafin). Biligrafin is not dependent upon an actively concentrating gallbladder mucosa in order to render the gallbladder radiopaque. Pheniodol enters the gallbladder and is not radiopaque; it becomes so only by the withdrawal of water. Telepaque is intermediate between these two.

To determine function, pheniodol is the best medium; to demonstrate nonopaque stones, telepaque; and to reveal anatomy, biligrafin. With these considerations in mind, the author outlines the basic plan that he uses as a routine for roentgenographic examination of the biliary tract. Following an interpretation of a wet roentgenogram of the entire abdomen and an interview by a radiologist, the patient is prepared accordingly. In general, the routine is similar to that used by most radiologists in this country.—*Frank J. Rigos, M.D.*

MILANÉS ALVAREZ, FERNANDO, CONDE OTERO, FRANCISCO, RODRÍGUES REMUS, CLEMENTE, EIBER HAFT, ENRIQUE, TRUJILLO IBARRA, CLARA AND HERNÁNDEZ MACHADO, JULIO. Valor de la bibligrafina intravenosa en el diagnóstico del síndrome de post-cholecistectomía. (Value of intravenous biligrafin in the diagnosis of post-cholecystectomy syndrome.) *Radiología, Panamá*, Sept., 1958, *8*, 153–160. (From: Hospital Universitario "Gral. Calixto Garcia," Habana, Cuba.)

The previously reported experiences regarding the use of cholografin in the post-cholecystectomy syndrome are confirmed in the results of 21 additional cases. Visualization of the biliary duct has been very satisfactory in a high percentage of cases, particularly in demonstrating the extrahepatic bile ducts. Visualization of the terminal portion of the common duct was enhanced by the use of such drugs as amyl nitrite and morphine. Some discrimination must be used in order to differentiate changes caused by functional and organic disease. Dilatation of the common duct in the post-cholecystectomy syndrome is a frequent finding.

In this series of cases, there was 100 per cent operative confirmation of the diagnosis made by intravenous cholangiography. Five of the cases are reported in some detail.—*Murray M. Friedman, M.D.*

BONI, ROBERTO. Cistifellea tripla e bile calcarea. (Triple gallbladder and calcareous bile.) *Radiol. med.*, Sept., 1958, *44*, 833–844. (From: Ospedale Civile, SS. Giacomo e Cristoforo, Massa, Italy.)

After briefly reviewing the importance of the roentgenologic examination for the visualization of congenital abnormalities of the gallbladder, the author reports a very unusual case.

A thirty-two year old woman was admitted to the hospital with symptoms related to a long standing gallbladder disease, at present in an acute phase. A preliminary roentgenogram of the right upper quadrant showed two radiopaque shadows, with the characteristic shape, size and position of two gallbladders un-united. The first impression of two gallbladders containing lime-milk bile was confirmed by an oral cholecystography which demonstrated also the presence of a third gallbladder, normal and functioning perfectly. An intravenous cholangiography demonstrated the presence of individual cystic ducts and of only one common bile duct.

The condition of the patient improved and she refused to undergo surgery. Although the roentgenologic findings are very conclusive, intervention would certainly have given more information on the morphology of the extrahepatic collecting system and permit a histo-pathologic study of this abnormality.

The author will present a further report in case the patient will decide to undergo surgery.—*A. F. Govoni, M.D.*

Gynecology and Obstetrics

Tetti, A., and Barbanti, A. Possibilità diagnostiche dell'amniografia. (The diagnostic possibilities of amniography.) *Radiol. med.*, Oct., 1958, *44*, 929–938. (From: Clinica Ostetrica e Ginecologica and Istituto di Radiologia, Università di Torino, Torino, Italy.)

In 1930, Menees, Miller and Holly (Am. J. Roentgenol. & Rad. Therapy, 1930, *24*, 363) were the first to propose the introduction of a radiopaque contrast medium into the amniotic sac. The method, however, was not widely accepted and it was only after recent research with water soluble contrast media, which are less toxic, that the procedure aroused new interest on the part of the radiologist and of the obstetrician as well.

The technique adopted by the authors is the following: after local anesthesia of the abdominal wall in the area selected, the needle, 10–15 cm. long and of No. 19–20 gauge, is introduced into the amniotic cavity. Fifty cc. of amniotic fluid are withdrawn and 20–40 cc. of a radiopaque contrast medium injected. Before taking any roentgenograms, the patient is asked to roll from one side to the other, in order to obtain a good spread of the contrast medium in the cavity. The most useful projections are the postero-anterior, the laterolateral and the obliques, each one chosen in accordance with the diagnostic problem and in order to reduce to a minimum the amount of radiation received by the fetus.

The indications for amniography are: (a) localization of the site of insertion of the placenta; (b) demonstration of abnormalities of the uterine cavity, of a possible mono-amniotic twin pregnancy, or of congenital malformations of the fetus; (c) determination of the sex; (d) confirmation of the presence of uterine contractions, the rupture of the membranes, or the fetal death.

In the opinion of the authors this procedure is innocuous and should be more widely adopted.—*A. F. Govoni, M.D.*

Breen, J. L., and Riva, H. L. Polyostotic fibrous dysplasia and pregnancy; a case report. *Am. J. Obst. & Gynec.*, Oct., 1958, *76*, 909–912. (From: The Obstetrics and Gynecology Service, Walter Reed Army Hospital, WRAMC, Washington, D.C.)

This case report is the second recorded instance of polyostotic fibrous dysplasia occurring as a complication of pregnancy, according to the authors. The diagnosis, although not established microscopically, was based upon the presence of *café au lait* spots on the right thigh, buttock and flank, lytic lesions of mutiple bones on the right side and an elevated alkaline phosphatase with normal serum calcium and inorganic phosphorus levels.

The pelvis was asymmetric, with cyst-like defects involving the ilium, ischium, pubis, and the right femur. A full term male infant was delivered vaginally. Immediate postpartum roentgenograms of the pelvis, in multiple projections, failed to disclose any evidence of pelvic fracture.—*J. H. Harris, Jr., M.D.*

Maurer, Hans-Joachim, and Wendt, Hermann. Zur Frage des Zusammenhanges zwischen Karzinomhäufigkeit und Geburtsmonat bzw. Blutgruppe. (The relationship of the incidence of carcinoma to the month-of-birth and to blood group.) *Strahlentherapie*, Sept., 1958, *107*, 144–154. (From: Strahleninstitut der Universitäts-Frauenklinik, Erlangen, Germany.)

A comparison of month-of-birth for 2,400 patients with carcinoma at the Erlangen Gynecologic Clinic, and for 2,400 control patients, showed no significant relationship between month-of-birth and the incidence of carcinoma. A brief summary of literature pertaining to the relationship between the incidence of carcinoma and blood group shows the correlation between these two factors is more likely than in the case of month-of-birth.—*Henry G. Moehring, M.D.*

Woltz, John H. E., Bradford, W. Z., Bradford, W. B., and McCoy, Joseph B., Jr. Complications of hysterosalpingography. *Am. J. Obst. & Gynec.*, Oct., 1958, *76*, 736–741. (From: Bradford Clinic, Charlotte, N. C.)

This is a report of the complications encountered in 500 consecutive hysterograms. A brief description of techniques is given. The complications were: (1) pain; (2) shock-like syndrome (nausea and vomiting, 2; fainting, 5; convulsions 2); (3) pelvic infection, 5;

(4) bleeding, 3; and (5) intravasation of material into the venous sinuses, 6.

None of the complications were serious. One patient who developed a pelvic inflammatory process was hospitalized.

The authors recommend that precautions be taken to perform this procedure when the patient's stomach is empty, when there is no known pelvic infection present and to discontinue the procedure upon the first sign of complications.—*J. H. Harris, M.D.*

Genitourinary System

Herman, John R., Mason, Brian, and Light, Igo. Fluorine in urinary tract calculi. *J. Urol.*, Oct., 1958, *80*, 263–268. (Address: J. R. Herman, 111 E. 88th St., New York 28, N. Y.)

Tissues and urinary calculi were obtained from 100 consecutive, unselected cases of urolithiasis. All of these patients lived at least part of their life in New York City, and they represented all races. There were 81 males and 19 females.

The calculi were washed with distilled water. Some stones were dissected so that the inner and outer layers were separated, others were ground *in toto*. Examinations were made for calcium, fluorine, phosphorus, ammonium, magnesium, oxalates, carbonates, and urates, according to the methods outlined in standard texts. Fluorine was determined quantitatively by the method of Willard and Winter. This involves fusion of the calculus with sodium carbonate, steam distillation of the fluorine as hydrosilicofluoric acid, and titration with thorium nitrate. Control studies showed that 92 per cent of fluorine was recovered by this method.

Fluorine content varied from nil to 1,800 parts per million. There was no correlation between the fluorine content of the stone and the sex, age or race of the patients. There was no correlation between the anatomic location of the calculus and the amount of fluorine it contained. Uric acid calculi contained only relatively small amounts of fluorine. Calculi containing 5 per cent or less of calcium showed fluorine in less than 100 parts per million. Calculi containing larger amounts of calcium frequently showed relatively larger amounts of fluorine, but there was no other direct correlation between these components. The fluorine present in the calculi bears roughly the same relationship to the phosphate content as it does to the calcium content. When there was less than 4 per cent phosphate, the fluorine was usually low and above 4 per cent phosphate, the fluorine was generally elevated above 100 parts per million.

In 38 surgical cases of calculus disease, tissue samples were taken for determination of fluorine content. There was no correlation between the amount of fluorine in the calculus and the amount in the tissues.

By roentgen-ray diffraction studies it was found that all calculi with a high fluorine content contained orthophosphates of calcium compounds known as apatites. Apatite was the only compound which was constantly found in all the high fluorine calculi. Apatite saturated with fluorine contains 3.77 per cent of fluorine. By calculation, the fluorine content of the stones analyzed in this study was generally below 0.3 per cent. The fluorine content of an apatite stone will vary in relation to the amount of fluorine in the urine, the length of exposure to the fluorine, the percentage of apatite in the calculus, and the surface area exposed. There is no reason at the present time to assume that the fluorine will increase the rate of growth of the calculus and no evidence is found to show that fluorine in the amounts ingested by the average population in the vicinity of New York has been toxic to the kidneys.

Two figures and four tables are presented in the analysis of the data obtained in this study.—*George W. Chamberlin, M.D.*

Marquardt, C. R., Pick, J. W., Melamed, A., Marck, A., and Holt, A. H. The use of miniature film (renogram) for kidney stone surgery. *J. Urol.*, Nov., 1958, *80*, 388–392. (Address: 759 N. Milwaukee St., Milwaukee 2, Wisc.)

The authors point out the value of roentgenographic examination of the exposed kidney during surgery for renal calculi. Various types of film examinations have been made. Now for the first time, there is available a factory prepared, custom designed film, contained in a light-tight plastic cassette. This cassette is $\frac{1}{16}$ of an inch thick and large enough to cover the average sized kidney. It can be sterilized in bichloride of mercury solution and it adapts itself well to molding to the kidney. Inherent in the design of this plastic cassette there is a notch along one border of the cassette which adapts itself to the renal pedicle. Opposite the notch, one corner of the film is cut obliquely to identify its location in relation to the kidney. The film is exposed and developed in the usual manner and the plastic cassette is disposable.

Numerous illustrations are presented to indicate the value of this type of roentgenographic examination.—*George W. Chamberlin, M.D.*

Dubilier, William, Jr., and Evans, John A. Peripelvic cysts of the kidney. *Radiology*, Sept., 1958, *71*, 404–408. (Address: J. A. Evans, 525 East 68th Street, New York 21, N.Y.)

Peripelvic cysts of the kidney are defined as cysts which are situated in the hilus of the kidney, intimately associated with the renal pelvis and calyces. They may be simple or multiloculated, single or multiple. These cysts are thought by some to be lymphatic cysts arising from the lymphatic struc-

tures in the hilus, probably a result of chronic inflammatory changes. Others believe they are congenital cysts arising from embryonic rests. These peripelvic cysts are relatively rare as there are few reported instances of this entity.

The authors report their findings in 13 cases of renal hilar masses demonstrated by pyelography in recent years. Surgical exploration has shown 7 of these to be peripelvic cysts and 2 lipomas of the renal hilus. Four have not been explored. All the cases showed about the same roentgenographic findings—a bizarre distortion of the renal pelvis. In three instances the mass was large enough to cause partial obstruction and caliectasis. Only one had sufficient obstruction to lead to a pyelonephritis with resultant symptoms. All the others had no symptoms which could be produced by the cysts. Although none of these patients had a renal hypertension the authors feel that, because these masses may cause this condition, they should be removed surgically.—*Donald N. Dysart, M.D.*

Ross, John A. Peri-ureteritis fibrosa, with notes on three cases. *J. Fac. Radiologists*, July, 1958, *9*, 142–146. (From: United Liverpool Hospitals, Liverpool, England.)

There is a disorder of the ureters and of the neighboring vessels, during the course of which they become embedded in fibrous tissue, the nature of which is uncertain. It is thought to be inflammatory in origin. The clinical symptoms and signs are not particularly typical of any disease condition. There is the complaint of vague, ill-defined backache, later perhaps becoming so severe as to prevent sleep.

Roentgenographic examination allows diagnosis in a relatively early stage. During the early part of the illness excretory urography may show normal form and function. Then as symptoms increase during a period of several months one may find gradually increasing nonprotein nitrogen, poor or nonvisualization of the urinary tract by intravenous urography, and difficult or impossible passage of contrast material on retrograde examination.

On operation the ureters are found embedded in firm fibrous tissue. This may completely enmesh a large part of the ureter, particularly from just below the renal pelvis downward to the level of the sacroiliac joints, with the lower part of the ureter much less enmeshed. After ureterolysis evidence of normal function is found on excretory urographic examination.

It is possible that cases may occur which may undergo a spontaneous cure, so that they may pass unsuspected owing to the vagueness of symptoms and signs. Other conditions suspected as a cause of the patient's complaints may be disk lesions and disease of the gallbladder. While this fibrous tissue formation has been said by some to be a response to some inflammatory lesion, the histologic appearance is not like that of an inflammatory process. Moreover, pyrexia does not usually occur during the course of the disease and enlarged lymph nodes are not found on exploration. Another suggestion has been that the disease may be initiated by periarteritis.—*Samuel G. Henderson, M.D.*

Peart, W. S., and Sutton, David. Renal-vein catheterisation and venography; a new technique. *Lancet*, Oct. 18, 1958, *2*, 817–818. (From: St. Mary's Hospital, London W.2, England.)

The usual technique for introducing a catheter into a renal vein has been to approach it from above through an arm vein. To enter the right renal vein in this way is a straightforward procedure, since the vein comes off the inferior vena cava at a downward angle. Catheterization of the left renal vein, however, is more difficult, because it comes off at a right angle to the inferior vena cava. Further, if admixture with blood from gonadal, suprarenal or lumbar veins is to be avoided, a catheter must be advanced right up to the kidney, since these veins join the renal vein in its proximal half. The authors devised a new technique whereby the bending of a catheter tip could be controlled during the observation under the roentgen-ray screen and the renal veins were approached from below.

The authors used a modified American nylon catheter, preferably a No. 8 and this was cut down to a 35 cm. length with usual syringe mountings at the end. A No. 5 nylon filament is threaded to an eyeless round-bodied needle and passed through the wall of the catheter 2½ cm. from the tip, passing from inside to outside. A single knot cut short is sufficient to secure. Next, the needle is passed through the wall into the lumen again 3 cm. below, and the thread is brought out of the bottom end of the catheter. The needle is then passed back through the wall of the catheter from inside to outside, just above the syringe mounting. The end of the thread is looped and it will then be found that traction on it will bend the tip in the required direction. Working the nylon thread in the holes will give the necessary sliding fit, so that the catheter can resume its natural shape when traction ceases. Care must be taken to have these holes through which the thread slides only a little larger than the thread in order to avoid leaks. These are usually very slight.

Under local anesthesia the saphenous vein is exposed below the saphenous ring; the catheter is introduced and passed upwards. Under roentgenoscopic guide the tip of the catheter is advanced to the level of the first lumbar vertebra and traction is applied on the thread so as to bend the tip and bring it up against the lateral wall of the inferior vena cava. When the catheter is pulled down gently, maintaining traction, the tip will usually spring into the mouth of the renal vein. The thread is then re-

leased and the tip of the catheter can be advanced along the renal vein. On the right, this would take it well into the flank. On the left, the tip usually stops at the left vertebral margin. During all these procedures normal saline containing 1,000 units of heparin per liter is dripped through the catheter. Blood can be drawn back rapidly, unless the tip is up against the vein wall, and in that case blood will flow if the catheter is gently withdrawn. The normal renal-vein blood can be recognized by its bright red color, compared with that in the inferior vena cava.

For venography the patient is instructed to practice the Valsalva maneuver. He performs this immediately before the injection of 15 ml. of 70 per cent diaginol or 70 per cent pyelosil. The contrast medium is injected as rapidly as possible, the aim being to deliver the total dose within two seconds. A film is exposed as the injected material reaches it. It is preferable to obtain several rapid serial films, rather than a single film, but with the screening table used by the authors, this was rather difficult and in practice they took a single film.

This procedure was carried out bilaterally in 27 patients, with a failure to enter one renal vein in 2 cases and a failure to enter either renal vein in 1 case.—*C. Peter Truog, M.D.*

Sproul, R. D., Fraser, Robert G., and MacKinnon, K. J. Aneurysm of the renal artery. *J. Canad. A. Radiologists*, Sept., 1958, *9*, 45–48. (From: Royal Victoria Hospital, Montreal, Quebec, Canada.)

Aneurysms of the renal artery will eventually rupture and cause death. Abeshouse states that 45 out of 115 cases reported in the literature died as a result of their aneurysms. Lowsley believes that most die within one year of the onset of symptoms. It is important, therefore, that this condition be diagnosed early so that appropriate and sometimes life-saving treatment may be instituted.

Clinically, the signs and symptoms are not characteristic. The diagnosis is made largely by roentgenologic studies. Calcification in the wall of the aneurysm is reported to be present in 25 to 50 per cent of cases. The shadow is a ring-like calcification overlying the medial upper third of the renal density, the outlines of which are deficient where it arises from the parent artery. Excretory and retrograde pyelography will demonstrate its close and constant relationship to the kidney. Aortography, whether calcium is present or not, will allow positive identification by opacification of the lumen of the aneurysm.

Two cases of renal aneurysm are reported. In each case the diagnosis was made preoperatively by the characteristic calcific shadow and aortography, and the lesion was successfully treated by nephrectomy.—*J. T. Ling, M.D.*

Spring, Maxwell, and Gross, Sidney W. Ruptured aneurysm of the circle of Willis associated with polycystic kidneys. *A.M.A. Arch. Int. Med.*, Nov., 1958, *102*, 806–810. (Address: S. W. Gross, 55 E. 82nd St., New York 28, N.Y.)

Aneurysms of the circle of Willis and polycystic disease of the kidneys are presumed to be the result of a congenital developmental defect. Both occur relatively infrequently. Their simultaneous occurrence in the same patient is more or less a medical curiosity. There are approximately 32 proved cases in the literature in which a combination of intracranial aneurysm and polycystic kidneys was noted.

The authors report a case of a forty-two year old female with a ruptured congenital intracranial aneurysm of the internal carotid artery associated with bilateral polycystic kidneys. The rupture of the aneurysm in the subarachnoid space and right frontal lobe resulted in alteration in cerebral function and meningeal irritation for which the patient was hospitalized. After the demonstration of the internal carotid artery by angiography, the common carotid artery was doubly ligated and divided. Recovery took place and the patient remained symptom free for four years and ten months and worked at her previous position. Signs of uremia developed and death occurred fifty-eight months after the subarachnoid bleeding. The autopsy findings are reported.—*C. Peter Truog, M.D.*

Whitley, Joseph E. A simple protection device for urologic units. *J. Urol.*, Oct., 1958, *80*, 277–278. (From: The Department of Radiology, the Bowman Gray School of Medicine, Winston-Salem 7, N.C.)

This simple device consists of a roentgenographic cone or double diaphragm inserted into the housing of the urologic roentgen-ray tube. This cone is made of $\frac{3}{16}$ inch steel, with a base plate of $\frac{1}{4}$ inch steel. The base opening measures a $1\frac{3}{16} \times 1\frac{7}{16}$ inches and the distal end $3\frac{1}{2} \times 4\frac{1}{2}$ inches. The vertical height is 6 inches. This cone is designed for a 36 inch target film distance and a mounting plate 3 inches below the target of the roentgen-ray tube. The design is such that it covers a 14×17 inch rectangular field at 36 inches.

After installing this cone, no measurable exposure was detected on the wrist or anterior thorax of the operator. The quality of the roentgenograms is not affected by this addition. One photograph of the rectangular cone over the Hugh Young unit and two diagrams illustrate the design of the cone.—*George W. Chamberlin, M.D.*

Nervous System

Psenner, L., and Schönbauer, E. Das Krankheitsbild der tuberösen Sklerose mit besonderer Berücksichtigung der röntgenologischen Symptomatik. (On tuberous sclerosis with special consideration of roentgenologic

symptomatology.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Sept., 1958, *89*, 301–318. (From: Zentral-Röntgeninstitut der Universität Wien im Allgemeinen Krankenhaus, Wien, Austria.)

Tuberous sclerosis is a familial hereditary disease with various manifestations in the central nervous system and skin. The classical triad of symptoms is idiocy, epilepsy, and acne rosacea, the latter also being known as adenoma sebaceum.

The family history usually reveals alcoholics and epileptics among the parents, with signs of degeneration and with various skin manifestations. The disease is discovered in infancy and childhood. Death usually occurs before the age of thirty either due to status epilepticus, increased intracranial pressure or intercurrent diseases.

The etiology is still unknown but, in view of associated spina bifida, testicular ectopia and/or other malformations, a developmental abnormality is believed to be the most likely cause. Pathologically, there are tuberous foci and small tumors of the heart and kidneys, and lesions of the brain, lungs, eyes, endocrine glands and skeleton. Histologically, the following findings have been reported: lipomyoma, angiolipoma, fibrolipomyoma and angiolipomyoma, all these occasionally with sarcomatous changes. Hypernephroma of the kidneys is a rare associated finding, likewise rhabdomyoma of the heart and presence of hamartomatous changes. The retinae may contain minute nodules; similar changes are occasionally found in the lungs.

Typical roentgenologic findings are disseminated intracranial calcifications, either homogeneous or granular, also areas of hyperostosis and osteosclerosis in the cranial vault. Rarefactions with cyst-like changes and osteoporosis may occasionally be found in the calvarium. Metacarpals and metatarsals can be the sites of lamellated periosteal proliferation and of sclerotic bone reaction. These changes simulate osteitis deformans, osteitis fibrosa, osteoblastic metastatic lesions, osteopetrosis, osteomyelosclerosis and osteitis multiplex cystoides. The lungs occasionally contain minute miliary densities.

Calcifying cerebral toxoplasmosis differs on the basis of more variability of foci with conglomerations, cluster formations and calcific streaks in addition to the calcified nodules.

A case of a twenty-nine year old female is reported with epilepsy, skin nodules, optic nerve atrophy and small foci in the retinae. Two years later the patient died in status epilepticus. Roentgenographic findings in addition to typical calcified intracerebral nodules were sclerotic and hyperostotic bone changes of the calvarium, also of the thoracic and cervical vertebrae, pelvic bones and of ribs. The tibiae and ulnae were the sites of symmetric cortical thickening and medullary narrowing. The metacarpals and metatarsals showed areas of endosteal and periosteal thickening. Cystic bone changes were noted in the distal phalanges of fingers and toes. Most of these skeletal changes have so far not been reported in the literature.

Numerous roentgenograms illustrate the typical features of diagnosis and differential diagnosis.—*Ernest Kraft, M.D.*

Skeletal System

Calandi, C., and Vichi, G. F. Studio clinico e radiologico su un caso di gargoilismo (malattia di Pfaundler-Hurler). (Clinical and radiologic study of a case of gargoylism [Pfaundler-Hurler disease].) *Riv. clin. pediat.*, July, 1958, *62*, 9–39. (From: Clinica Pediatrica and Istituto di Radiologia dell'Università, Firenze, Italy.)

The authors had the opportunity to study clinically and roentgenologically an infant affected by gargoylism. This study was made at the age of forty days and of seventeen months.

On the first admission, the roentgenologic, clinical and laboratory findings were such as to conclude for the presence of a chondro-osteodystrophy of unknown etiology. It is only during the second admission that clinical, hematologic and roentgenologic studies demonstrated the characteristic features associated with the disease of Pfaundler-Hurler.

The etiology, the pathologic, clinical and roentgenologic changes of gargoylism are reviewed.

The article is well illustrated and an extensive up-to-date list of references is added.—*A. F. Govoni, M.D.*

Broder, Harold M. Prognosis in Legg-Perthes disease. *J. Pediat.*, Oct., 1958, *53*, 451–463. (From: Department of Orthopedic Surgery, University of Pennsylvania Hospital, Philadelphia, Pa.)

This article reviews 208 cases of Legg-Perthes disease regarding prognosis using Heyman and Herndon's "comprehensive quotient" to measure results.

Males represented 79.8 per cent of the cases. The age of the patients ranged from 2.8 to 14 years with about half of the cases falling between six and nine years of age. Right-sided involvement was found in 46.4 per cent, left-sided involvement in 40.6 per cent, and bilateral involvement in 13 per cent.

It was found in the patients studied that the age, severity of involvement, and status of progression after treatment was begun had a direct bearing on the final results.

The prognosis is best in the younger age group, when the involvement is less severe, and treatment is started early.

The author found no advantage of bedrest with or without traction over the ambulatory non-weight bearing methods except in cases of bilateral involvement.—*George A. Miller, M.D.*

Stanisavljevic, Stanko, and St. John, Elmer G. Congenital fusion of three lumbar vertebral bodies. *Radiology*, Sept., 1958, *71*, 425–427. (Address: S. Stanisavljevic, The Henry Ford Hospital, Detroit 2, Mich.)

Congenital fusion of cervical vertebral bodies is fairly common and of thoracic vertebrae not too uncommon. Such involvement of lumbar vertebrae is rare, with few cases recorded in the literature.

The authors report a case in which fusion of the the first, second, and third lumbar vertebral bodies was found. There was also fusion of the eighth and ninth ribs on the right side and a rudimentary eighth thoracic vertebral body. The roentgenographic reproductions, produced by the LogEtronic method, illustrate these findings.—*Donald N. Dysart, M.D.*

Rezek, J. Die Arthrographie. (On arthrography.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Sept., 1958, *89*, 319–331. (From: Radiologische Universitätsklinik, Prag, Czechoslovakia.)

The value of arthrography continues to be disputed although more than fifty years have elapsed since introduction of the method. The author has gained valuable information from the procedure and has not experienced any complications in a seires of almost 1,000 cases. The only contraindications are acute inflammatory processes and hypersensitivity to iodine. It is stated that considerable experience is essential for the proper interpretation of arthrograms.

The *knee* is the joint most frequently subjected to arthrography. In a series of 800 cases 67 per cent have been found abnormal. Rupture of capsule, dislocation, subluxation and fracture of menisci, also meniscectomy with regeneration and synovitis have been the most frequent findings. Pneumoarthrography with double contrast has been used in 100 cases, but instead of yielding additional information it has shown less details than simple arthrography with contrast material. Three illustrations show chronic synovitis, sclerosis of the joint capsule and meniscus degeneration, respectively.

The *temporomandibular joint* has been studied in 24 cases. Only 1 or 2 cc. of iodine solution need be injected. Arthrography of the joint is warranted when ordinary roentgenograms and laminagrams are not revealing, but is contraindicated in fractures of the mandibular head because of associated tear of the joint capsule. The method helps to discover meniscus lesions and capsule distention of long standing.

The *shoulder joint* is injected on the horizontal table under fluoroscopic control. Injection of 8 to 10 cc. of an iodine solution can be easily tolerated. Pain is usually a sign of para-articular injection. The arthrograms may show capsule and tendon rupture, especially in the supraspinatus area, and dislocation of the shoulder with close approximation of contrast material to the greater tuberosity of the humerus. In frozen shoulders the method is of no value.

The *elbow joint* is injected posteriorly from above into the olecranon fossa. An amount of 5 to 6 cc. is sufficient for proper studies. Damage to ligaments and joint capsule can be demonstrated, and the method is simple and helpful to the surgeons preoperatively.

The *ankle joint* is injected anteriorly toward the medial malleolus. The method has been used in 62 fresh injury cases without causing complications. Typical indications for the arthrography are severe traumatic tissue damage, checking of joint capsule and post-traumatic disturbances. Cases are illustrated showing tears of ligaments, of the joint capsule and of the tibiofibular syndesmosis. The extent of a hematoma also can be shown with this method.

It is concluded that arthrography is simple and is indicated in all cases in which the clinician cannot make a definite diagnosis and, therefore, desires additional information.—*Ernest Kraft, M.D.*

Osserman, Elliott F. Natural history of multiple myeloma before radiological evidence o disease. Memorial Fund Lecture. *Radiology*, Aug., 1958, *71*, 157–174. (Address: 99 Fort Washington Ave., New York 32, N.Y.)

The author describes recent clinical and laboratory studies which support the thesis that multiple myeloma which can be demonstrated roentgenographically has already progressed to an advanced stage. Plasma cell proliferation may exist for long periods prior to the appearance of clinical signs or symptoms. It is frequently possible to make the diagnosis by biochemical techniques before roentgen changes are demonstrable.

A group of 24 patients whose sera were discovered to contain an abnormal protein of the myeloma type, but who displayed neither symptomatic nor roentgenographic evidence of osseous disease, were studied by the author. In all instances the demonstration by electrophoresis of a characteristic abnormality in the serum proteins indicated the presence of a primary plasma-cell proliferative disorder. All patients were observed for periods ranging from a few months to over three years. In two patients skeletal damage and disease patterns consistent with a diagnosis of myelomatosis developed. Necropsy examination performed on two patients, revealed diffuse plasma-cell infiltration of the skeletal system. Eighteen patients continue under careful clinical and laboratory scrutiny.

A combination of several laboratory and clinical techniques is needed to discover and evaluate cases of plasma-cell proliferative disease. Routine urinary protein electrophoresis in patients with unexplained proteinuria may identify some early cases. Routine use of the serum protein electrophoresis test should

result in diagnosis of this disease in the earlier stages, although the test has definite limitations with regard to its ability to distinguish small amounts of abnormal protein.

Clinical and laboratory data of the 24 patients in this series are presented in detail. Filter paper serum electrophoretic patterns of all cases are reproduced. —*Arno W. Sommer, M.D.*

Blood and Lymph System

Gerard, Franklyn P., and Lyons, Harold A. Anomalous artery in intralobar bronchopulmonary sequestration; report of two cases demonstrated by angiography. *New England J. Med.*, Oct. 2, 1958, *259*, 662–666. (From: The Department of Medicine, State University of New York, Downstate Medical Center, and the Department of Thoracic Surgery, Division I, Kings County Hospital Center, Brooklyn, N.Y.)

Intralobar bronchopulmonary sequestration of the lung refers to the association of a congenital cyst of the lower lobe of the lung with an anomalous artery arising from the aorta either above or below the diaphragm. The main etiologic factor is the anomalous artery. It is this artery which, if the condition is not recognized, leads to serious operative mishaps and death. For this reason, angiographic examination should become a part of the diagnostic procedure in every suspected case of this condition. The authors show the value of preoperative demonstration of an anomalous vessel in 2 patients with an intralobar bronchopulmonary sequestration. The diagnosis of an aberrant artery supplying the pulmonary area was established in each case by angiography.

The first was a case of a fifty-eight year old Puerto Rican in which a persistent wedge-shaped area of increased density at the right cardiophrenic angle lying posteriorly was demonstrated. Angiocardiographic study revealed an anomalous systemic blood vessel arising from the abdominal aorta, perforating the diaphragm to supply the density. In addition to the abnormal lung tissue a herniated portion of the liver was also found. Both the diseased lung and the herniated portion of the liver were removed.

The second case was a six-and-a half year old Puerto Rican girl. A plain roentgenogram showed a density in the left lower lung field surrounded by an area of pneumonitis. In the lateral view a lobular density in the left posterior basilar segment was noted. Aortography was performed and this demonstrated one large and several small anomalous vessels arising from the thoracic aorta that supplied the sequestrated area of the left lower lobe. Thoracotomy was carried out and the infected pulmonary segment was removed. Both of these patients made an uneventful recovery.

In their summary the authors plead for the angiographic preoperative demonstration of the anomalous vessels to confirm the diagnosis and allow the proper planning of surgery.—*C. Peter Truog, M.D.*

Rösch, J., Bret, J., and Lišková, M. Die Splenoportografie in der Diagnostik der Splenomegalie. (Splenoportography in the diagnosis of splenomegaly.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Sept., 1958, *89*, 249–268. (Address: Josef Rösch, V. Borcové 2, Praha-Břevnov, Czechoslovakia.)

The cause of splenomegaly can be clinically ascertained in most instances; but in a few cases with obscure etiology splenoportography has proved to be very helpful.

The authors have modified the technique of splenoportography in the following way: the injection of contrast material is done under fluoroscopic control on the horizontal table, and the patient stops breathing in the inspiratory phase for the duration of the examination. The latter usually can be expedited during a twenty-seconds' period, but the patient has to be carefully trained for the required cooperation. No complications have followed this type of technique.

Four hundred and fifty cases have been studied, and in 50 per cent the diagnosis has been verified by operation and/or autopsy. Failure due to incomplete filling has only occurred in eleven patients (2.5 per cent).

The first group of cases comprises systemic splenomegalies in leukemia, polycytemia, lymphogranuloma, hemolytic icterus, Gaucher's disease, Niemann-Pick's disease and infectious diseases. There is dilatation and tortuosity of the splenic vein, portal vein and large hepatic veins, normal circulation time, low saturation of contrast material and absence of collateral circulation. The larger the spleen, the more pronounced are these changes.

The second group includes splenomegalies due to extrahepatic block of the portal vein. The three causes are either a congenital anomaly, thrombosis or extrinsic pressure. Collateral circulation develops early and can easily be proved by splenoportography. Other findings are nonfilling at the site of the block, prestenotic dilatation of veins, decreased circulation time, and deviation of veins in case of extrinsic pressure.

The third group of splenomegalies consists of underlying liver disease due to hepatitis or cirrhosis. One hundred and fifty cases have been studied in this group. In hepatitis the intrahepatic veins are thin and sparse. The small branches show an irregular pattern with absent filling peripherally. In hepatic cirrhosis these changes are still more pronounced. Vascular filling is only noted centrally, while the periphery of the liver appears avascular. This picture has been likened to an old dried out tree with irregu-

lar branches radiating toward the periphery. Collateral circulation depends on the degree of damage to the hepatic flow with central stagnation.

In neoplasm and trauma to the spleen there is very little gained from splenoportography.

Seventeen roentgenograms illustrate the described findings.—*Ernest Kraft, M.D.*

MISCELLANEOUS

Gerstner, Herbert B. Acute clinical effects of penetrating nuclear radiation. *J.A.M.A.*, Sept. 27, 1958, *168*, 381–388. (Address: Route 3, Box 512 C., San Antonio, Tex.)

All penetrating ionizing radiations, regardless of physical nature and origin, are uniform and comparable in biologic effect. Ionizing radiation temporarily inhibits mitosis in sensitive cell systems with high proliferative activity. The organism continues its normal function until the store of mature critical cells is exhausted and then the disturbance becomes overt because of deficiency of new cell supply. Since mitotic damage is transitory, normal function will be resumed if the organism is able to survive the critical period of maximal cell depression.

In an attempt to predict type, degree, time course, and incidence of disease resulting from acute radiation, four sources of information have been used: Atomic bomb casualties, nuclear accidents, radiotherapy, and animal experiments.

A transitory systemic reaction of nonspecific character occurs within a few hours of exposure, before any of the specific type reaction. This type of systemic response varies for the species of animal, and the severity varies greatly in individuals of the same species. In man, the symptoms of the systemic reaction resemble seasickness or airsickness, and vary from light nausea in some to severe distress requiring sedation in others; but have usually subsided completely by the third day.

The different organs show a wide diversity with respect to sensitivity, and the clinical picture is characterized by that affected essential organ which has the shortest latent period. The three organs which appear to be important in considering acute clinical effects are the central nervous system, small intestine, and bone marrow together with lymphoid tissue. To trigger acute clinical sequelae in the central nervous system a dose of several thousand roentgens is required, and this acute cerebral damage reveals itself within a few hours, while injury to other organs is still latent. There is rapid transition through languor, apathy, prostration, and, with doses in excess of 5,000 r, tremors, convulsions, ataxia and death. The morphologic changes responsible are probably nonbacterial inflammatory infiltrations which appear throughout the central nervous system producing radiation vasculitis, meningitis, and encephalitis, followed by a disturbance in the blood-brain barrier leading to brain edema

For the small intestine the threshold dose is around 500 r, and the latent period approximately five days. The onset of symptoms is rather abrupt, with nausea, vomiting, malaise, fever and diarrhea. During the second week after exposure, dehydration and hemoconcentration develop, and finally coma followed by death in circulatory collapse. These symptoms are the result of denudation of mucosa of the duodenum and jejunum. Mucosal epithelium in the intestinal tract is normally sloughed off, but the radiation inhibition or arrest of mitoses interferes with the normal replacement of this tissue. The denudation is followed by infection and its consequences. Therapy is based on maintaining fluid and electrolyte balance, and on antibiotics.

For the hematopoietic system the threshold dose is approximately 100 r and the latent period about three weeks. After the prodromal symptoms subside, the patient has a symptom free period of about two and a half weeks, at which time, approximately nineteen to twenty days after irradiation, he experiences chills, fever, headache, and dyspnea on exertion. Within a few days sore throat, pharyngitis, bleeding gums and petechiae coincide with the agranulocytic blood picture. Convalescence begins about the end of the second month and is usually fairly complete in another month.

The three forms of acute radiation syndrome are not as completely dependent as described, but usually are seen as a mixture of two adjacent forms, particularly of the gastrointestinal and hematopoietic forms. The latter will probably pose the major problem facing the physician under catastrophic conditions. Realizing that quoted values are only guidelines, doses less than 100 r can be considered of no acute concern, and doses in the range of 200 r may be considered threshold hospitalization level.—*Lois Cowan Collins, M.D.*